(11) ANON: World action on smoking. Brit Med J 4: 65, 1971

Carbon monovide may be a toxic ingredient of tobacco smoke that deserves mere attention than it has received. P. Astroph has recently reported that exposure of rabbits to low concentrations of carbon monoxide can lead to production of atteroism. The consequences of the blood of eigerctre smokers rany exceed 10%, and Astrop believes that this may be more important than pleating in relation to coronary disease. Further research on this aspect is certainly headed.

There are two statements that should be modified. Astrup's experiments were performed on rabbits fed with cholesterol and their blood levels showed 15% carboxyhemoglobin (see page 69). That the blood levels of smokers may exceed 10% is a rarity (see pages 20-21).

(12) ANON: Cigarette smoking and carbon monoxide. Mcd Letter Drug Ther 13: 91-2, 1971.

In addition to nicotine, tars, and other chemical compounds, carbon monoxide has been incriminated as a pathogenic factor in cigarette smoke. Recent studies have suggested that heavy cigarette smoking (more than 20 cigarettes a day) may result in an intake of carbon monoxide that could impair the performance of the smoker in driving a car or piloting an airplane.

The average concentration of carbon monoxide in cigarette smoke is about 20,000 parts per million or about 400 ppm in the inhaled mixture of smoke and air (J. R. Goldsmith and S. A. Landaw, Science, 162:1352, 1968). The additive effects of carbon-monoxide-polluted air must also be taken into account. In Los Angeles, where high atmospheric carbon monoxide levels have caused concern, the concentration in the air during a four-year study ranged from 7.3 to 20.2 ppm (A. C. Hexter and J. R. Goldsmith, Science, 172:265, 1971).

The reference to the paper by Goldsmith and Landaw (1968) regarding 400 ppm of an inhaled mixture of smoke and air appears in this article. Since Goldsmith and Landaw cite no reference, it is not possible to challenge their source. Jongbloed (1939) analyzed the alveolar air and noted a peak level of 31.5 ppm carbon monoxide. Ringold et al (1962) analyzed the expired air after a 20-second breath-holding period and noted a level of 16.4 ppm for heavy smokers (see page 19).

- (13) ARONOW W S, KAPLAN M A and JACOB D: Tobacco: A precipitating factor in angina pecteris. Ann Int Med 69: 529-36, 1938.
- (14) ARONOW W.S., DENDINGER J and ROKAW S.N.: Heart rate and carbon monomide level after smoking high-, low-, and non-nicotine cigarettes. A study in male patients with angina pectoris. Ann Int. Med. 74: 697-702, 1971.
- (15) ARONOW W S and ROKAW S N: Nonnicotine cigarettes. Effects in angina pectoris. Circulation 44: 782-8, 1971.
- (16) ARONOW W S, HARRIS C N, ISBELL M W, ROKAW S N and IMPARATO B: Effect of freeway travel on angina pectoris. Ann Int Med 77: 669-76, 1972.

These four publications from Aronow's group have been widely quoted as supporting the theory that carbon monoxide causes coronary heart disease. It should be noted that the investigation concerns carbon monoxide contained in cigarette smoke and in vehicular exhaust. There is no comparative study using carbon monoxide in air to ascertain that the results are due to carbon monoxide contained in vehicular exhaust or cigarette smoke.

Not all four reports include blood analysis for carboxyhemoglobin. In the first, none is recorded, in the second, blood was analysed, in the third, alveolar-expired air was used, and in the fourth, both blood and air had analysis.

- (17) AYRES S.M., GIANNELLIS Jr and MUELLER II: Myocardial and systemic responses to carboxyhemoglobin. Ann NY Acad Sci. 174: 268-93, 1970.
- (18) AYRES S M, MUELLER H S, GREGORY J J, GIANNELLI S Jr and PENNY J L: Systemic and myocardial homodynamic responses to relatively small concentrations of carboxyhemoglobia (COHB). Arch Environ Health 18: 699-709, 1969.

These 2 papers have been widely quoted as important evidence of the role of carbon monoxide in causation of coronary heart disease. It is important to examine the methods used which have a basic fault, viz, inhalation of 5% carbon monoxide, producing blood levels of 9%. Other investigators have attained 50% to 90% carbony-hemoglobin levels with inhalation of 5% carbon monoxide. The blood samples were drawn by Ayres et al 10 minutes after starting the inhalations, which means that the blood had not reached equilibrium with the alveolar air. The observations by Ayres et al were on a nonsteady state. It is customary to measure coronary blood flow and myocardial metabolism during a steady state of at least 10 minutes.

Methods

Studies were performed on 26 patients during routine diagnostic cardiac catheferization and in 31 dogs. The patients were studied in a cardiopulmonary laboratory following completion of diagnostic procedures. Catheters were placed in the ascending north and pulmenary artery. A Geodale-Eulin outheter was placed in the proximal coronary sinus in 12 of the human studies and in 16 of the conine studies. Mixed expired air, arterial, mixed venous and coronary sinus bloo I was obtained prior to administration of carbon monoxide. The subject was then switched to a minture of mercarbon monoxide in air for from 35-to 120 seconds, with continual electrocardiographic monitoring. Following a ten-minute period to permit washout of alveolar carlon monoxide, repeat blood same ples and mixed expired air were collected while breathing room air. Coronary blood flow was measured immediately before and ten minutes after carlon monoside breathing.

The carboxyhemoglobin levels after exposure to 0.01% to 1.0% is as follows:

CORRELATIONS OF ATMOSPHERIC CARBON MONOXIDE EMPOSURE AND CARBONYHEMOGLOBIN				
CO IN AIR (PER CENT)	Duration of Enfosure (80% equilibrium)	COIIn (per cent of elood-CO saturation)		
*0.01 (109 ppin) 0.02 to 0.03 0.04 to 0.03 *0.1 (1090 ppm) 0.2 0.3 0.4 0.5 to 1.0	6 or more hours 5 to 6 hours 4 to 5 hours 3 or more hours 1 hour 30 minutes 20 to 30 minutes 2 to 15 minutes	17 23 to 30 36 to 44 50 55 to 60 50 68 75		
how long the exposure	encentration of 0.01% leads to CO concentration of 0.1% leads to 2 CO are. One half of the available h	otto an accordance of the		

(19) AYRES S M: Roles of carbon monoxide and nicotine in circulatory effects of cigarette smoke. JAMA 219: 520, 1972.

> Roles of Carbon Monoxide and Nicotine in Circulatory Effects of Cigarett Smoke

I would like information about the specific factors that cause cigarette smoking to have an adverse effect on the cardiovascular system. Is it only nicotine that is involved, or can carbon monoxide and its effect on carboxyhemoglobin levels be implicated?

Until recently, the pharmacology of tobacco smoke was thought to be essentially that of nicotine, and most early articles on the toxicity of cigarette smoke emphasized nicotine to the exclusion of other considerations. It seems likely, however, that the 3% to 4% of carbon monoxide found in cigarette smoke may play an important role intensifying the recognized cardiovascular toxicity of nicotine. Carboxyhemogiobin levels as low as 3% to 4% may increase the oxygen debt of exercise and we have shown that levels of carboxyliemoglobin between five and ten percent may produce abnormal myocardial metabolism in patients with coronary artery disease. Smokers generally have 3% to 7% of their hemoglobin saturated with carbon monoxide. The whole subject has been recently reviewed in a New York Academy of Science monographion carbon monoxide² and in the progress reports from the Surgeon General's office on eigarette smoking and health.

The toxicity of tobacco smoke appears to derive from both its nicotine and carbon monoxide content. Nicotine increases cardiac work by increasing heart rate and blood pressure: Carbon monoxide interferes with the ability of the heart to extract oxygen from the perfusing blood. The combination of increased oxygen requirements and decreased oxygen availability may well lead to myocardial ischemia, particularly in patients with coronary artery disease.

St. Vincent's Hespital and Medical Center

^{1.} Chevalier RB, Krumholz RA, Ross JC: Effects of carbon monoxide inhalation on the cardiopulmonary responses of non-smokers to exercise, J Lab. Clin. Med. 62:167, 1963,

^{2.} Culmen RF (ed): Riological Effects of Carbon Monacidi, New York, New York Academy of Science, 1970.

The author cites Chevalier et al, a reference which appeared in 1963 as an abstract and does not comain results of blood levels of carboxyhemoglobin. In a subsequent paper (JAMA 198: 1061-64, 1966) Chevalier et al reported the results of inhaling 0.15% carbon monoxide, which caused a carboxyhemoglobin level of 3.95%. This value is lower than results of others, who obtained levels of 10% to 15% of carboxyhemoglobin. Chevalier et al used an indirect technique based on analysis of alveolar air, so that it is possible that by this technique they underestimated the true value if blood analysis was used. The technique employed was as follows:

For determination of COHb levels in the blood, the relationship of Haldane and Smith was used. Forster and co-workers, adapted the Haldane relationship for determination of COHb by using a modification of Sjöstrand in which alveolar gas is equilibrated with pulmonary blood and the equilibrated capillary carbon monoxide tension is measured directly in a sample of expired alveolar gas. This method, as utilized by Forster and co-workers, and gave results in the same range as those found by analysis of whole venous blood for COHb. Our technique for equilibration of capillary carbon monoxide tension was similar to that described above with certain modifications. The Haldane relation states the following (atm signifies atmosphere):

COHb/O.Hb = 210 CO pressure in atm/O. pressure in atm

% COFIb =
$$\frac{O_2 \text{ pressure in atm}}{210 \text{ CO pressure in atm}} + 1$$

When there is little reduced hemoglobin present, 210 is the M fraction established by Haldane. This technique for determination of COHb showed a satisfactory relationship when compared with the COHb level as determined spectrophotometrically on the venous blood of nine control subjects.

One hundred million motor vehicles driven currently in the United States and well over 50% billion cigarettes smoked in this country annually represent a vicious combination of machine-made and self-inflicted air pollution. Most frequent victims of this nationwide health hazard are heavy smokers accustomed to driving a car or other

motor vehicle as part of their daily design for living. High temperature and humidity of the atmosphere aid rapid uptake of carbon monoxide by hemoglobin. Also, hyperventilation increases carbon monoxide uptake and decreases its removal from the blood. In subjects with emphysema, absorption of carbon monoxide is reduced because not all of the tidal air is reaching the pulmonary capillaries. In urban population, normal blood contains a small amount of carbon monoxide: 0.62-1.24 percent as earboxyhemoglobin. The affinity of hemoglobin for carbon monoxide is 250 times greater than for oxygen. Relative to the cardiovascular system, it has been noted in experimental animals that exposure to toxic concentrations of carbon monoxide brings about progressive increase in the pulmonary artery pressure, increased heart rate and systolic pressure, together with decreased metabolism. Blood pressure decreases when carboxyliemoglobin values of the blood reach 10 percent. Moreover, intermittent exposure to toxic amounts of or actual poisoning with carbon monoxide may be associated with reversible inversion of T wave, elevation of R-T segment, atrioventricular dissociation and A-V block. These changes, as well as associated degenerative sequels insome of the myocardial fibers, small hemourhages in and necrosis of the myocardium resemble closely alterations due to severe hypoxia from other causes. In humans suffering from coronary heart disease, the extraction of oxygen from the blood by the myocardium is reduced at carboxyhemoglobin levels between 5 and 12 percent. This phenomenon may be a contributory factor to the increased incidence of augina pectoris and coronary thrombosis in the individuals involved. Another adverse result of carbon monoxide is increased viscosity of the blood. Rabbits inhaling 6:017 percent carbon monoxide in air for several weeks develop atheroselerosis and myocardial necrosis. Atherematous changes are pronounced in cholesterel-fed animals inhaling low concentrations of carbon monoxide as compared with cholesterol-fled animals without exposure to carbon monoxide. Astum et al (Confere ence on Biologic Effects of Carbon Monoxide, New York Acad Med, 1969); observed in animal experiments that carbon monoxide rendered vessel walls more permeable, with consequent increased flow and deposition of fat in vessel walls and development of atherosclerosis. Observations in humans show that low concentration of carbon monoxide may lead to inhibition of bioelectric activity of the brain. Impairment in cognitive and psychomotor areas of the brain may be associated with earboxylemoglobin levels between 2 and 5 percent. In the lung, hypoxia caused by earbon monoxide inhibits the function of alveolar macrophages. This, in turn, weakens tissue defense against airborne bacterial infection. An apropos question my be posed: if the recurrent or sustained by

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poxic state of the lung tissue might not favor development of bronchogenic carcinoma under the influence of carcinogens of cigarette smoke. One hundred million

Commentary (20) Banyai, Cont.

motor vehicles discharge 66 million tons of carbon monoside anamally. No wonder that in all major cities at busy intersections during hours of peak traffic the concentration of this hammful gas is much higher than the maximum allowable concentration, victimizing drivers, pedestrians and traffic policement. Carbon monoside in cigarette smake is an incomplete combustion product even though the temperature at the burning zone of the cigarette is 854°C (1,655,2°H) white air is being drawn through the eigarette. Carboxybemoglobin level of the blood is 446 percent immoderate smokers and up to 12 percent in heavy smokers. Its potential hazard can be estimated by adding these figures to those pertaining to motor vehicle drivers.

Andrew L. Banyai, M.D.

- a. If hyperventilation increases carbon monoxide uptake, it will also hasten its removal from the blood. One technique for promoting the elimination of carbon monoxide in acute poisoning is to increase depth of respiration by having the patient inhale 5% carbon dioxide in oxygen.
- b. In urban populations, the carboxyhemoglobin level in the blood is greater than 0.62% to 1.24%. In the 26 investigations reported in the literature the overall mean for 1,662 subjects was 1.45%. Residents of London, Los Angeles and Milan show the following mean levels respectively: 3.5%, 2.3% and 2.8%. These values represent a significant contribution by carbon monoxide to pollution in the atmosphere (see pages 32 and 33).
- c. The effects listed in 15 lines are derived from toxic concentrations of carboxyhemoglobin ranging from 50% to 100%. The phrase "toxic concentration" appear only in the first sentence but applies to the next 3 sentences.
- d. The experiments in rabbits fed with cholesterol are not supported by those in dogs reported by De Bias et al (1972). Chronic exposure to carbon monoxide does not exaggerate myocardial ischemia (see page 62).
- e. There is no published report that psychometer and cognitive areas in the brain can be influenced by levels of carboxyhemoglobin between 2% and 5%. More accurate statistics would be between 5% and 20% (see pages 88-89).
- f. The effect of carbon monoxide on alveolar macrophages of experimental animals is encountered with levels of 0.5% to 2% in inspired air. Smokers have a level of carbon monoxide of 7 to 12 ppm in expired air, which is 1000 less than the concentration used in experiments on animals.
- g. The levels of 4% to 6% carboxyhemoglobin in moderate smokers and 12% in heavy smokers is not supported by values stated in the literature. Balbo et all (1966) reported a mean level of 2.8% for 7 smokers, each consuming 30 cigarettes daily. Rouch et al (1971) reported a mean of 4.25% for 15 smokers using more than 10 cigarettes daily (see page 15).

Source: https://www.industrydocuments.ucsf.edu/docs/yfmk0000

(21) BARTLETT D fr: Pathophysiology of exposure to low concentrations of carbon monoxide. Arch Environ Health 16: 719-27, 1968.

Regular eighrette smokers have repeatedly been shown to have COED concentrations in the 5%, to 30% range of Smokers of pieces and cigars have COIIb levels that are somewhat lower than those of cigarette smokers. but higher than those of nonsinokers. These findings have led to the widespread error of supposing that smokers may be mere susceptible to environmental CO than nonsmokers. Carbon monoxide from eigarette smoke and CO in the ambient air are not. additive in their biologic effect. Carbon monoxide is absorbed only when the Pco in the ambient air exceeds that in the pulmonary capillary blood. Thus, persons with COHb levels of 5% from smoking do not absorb further CO from the environment unless the ambient CO concentration is 30 ppm or more; on the contrary, they excrete CO at a rate roughly proportional to the Pco gradient between their blood and the ambient air. This suggests that smokers may be among the least susceptible of persons exposed to low atmospheric concentrations of CO, since their COHb concentrations are not increased by the exposure. This conclusion is modified, however, by the fact that smokers' CO exerction between cigarettes is slower in a CO-polluted environment than in pure air. Thus, their long-term average COHb concentrations are slightly highly in the presence of environmental CO than in its absence.

heart disease is considerably higher for smokers than for nonsmokers. The rate for examokers is no higher than for persons who have never smoked. This pattern implies that the smoking effect is completely reversible when an individual stops smoking. Thus, smoking must cause myocardial hypoxia by some acute, reversible precess, probably unrelated to the formation of hard, irreversible, atheroselerotic lesions. Carbon monoxide fits this epidemiologic pattern quite well, but nicotine or other components of cigarette smoke may be responsible, and the question remains unsolved.

The two paragraphs quoted from this article emphasize two points: (1) Carbon monoxide from cigarette smoke and that in the ambient air are not additive in their biologic effect; and (2) the effect of smoking on coronary heart disease is reversible. These two points were missed in the main text of this review and their conception properly belongs to Bartlett.

(22) CAMM A J: The effects of smoking. Guy Hosp Gaz 81: 185-203, 1967.

Some three hundred different constituents of tobacco smoke have been identified, many of them in infinitessimally small quantities. The two substances present in the greatest amounts are carbon monoxide and nicetine. Although carbon monoxide is found in high proportions in the mainstream smoke of a cigarette, it is seldom found in high proportions combined with haemoglobin in the blood. The percentage of carbony-haemoglobin rarely rises above five per cent unless cigarettes are "chain-smoked" in which case it may rise to ten per cent. This is not sufficient to be of clinical significance.

This review article includes a section on "contents of tobacco smoke". The paragraph quoted summarizes the present status of carboxyhemoglobin levels in cigarette smokers, concluding with the statement: "This is not sufficient to be of clinical significance".

(23) CONROY J P: Smoking and the anesthetic risk. Anest Anal 48: 388-400, 1969.

It takes 24 hours in a carbon monoxide free atmosphere to reduce a carboxyhemoglobin of 18 to 5 percent. Is it entravegant to demand 72 smoking-free hours before anesthesia?

The 24 hours required to reduce carboxyhemoglobin in the blood from 18% does not apply to the time required to reduce from 5% the blood level of carboxyhemoglobin in cigarette smokers. In the literature, 4 to 12 hours after smoking, the mean blood level for 2,054 habitual smokers is 3.76%. This value, compared with the level for nonsmokers, represents an increase of 2.19% attributed to smoking. Because of endogenous and exogenous sources of carbon monoxide other than cigarette smoking, it is not possible to reduce the carboxyhemoglobin level below 1.5% (see pages 12-13). An answer to the question raised by Conroy should be as follows: Waiting 72 hours is unreasonable; 4 to 12 hours would be sufficient.

(24) CURPHEY TJ: Carboxyhemoglobin in relation to smoking. Nat Cancer Inst Monogr 28: 231-5, 1968.

This is an excellent discussion of the significance of the carboxyhemoglobin level in relation to smoking. The entire article is approduced.

Carbonyhemoglobin in Relation to Snicking

THEODORE J. CURRIER, M.D., Chief Medical Examiner—Coroner, Los Angeles County, Los Angeles, California 90012

HE main thrust of the Conference and the tenor of discussion in the general sessions and in this workshop have been to review and analyze the various agents in tobacco smoke with regard to their potential threat to the health and well-being of the eigerette smoker. The evidence already presented has dealt largely with those effects of certain components of tobacco smoke as they relate to such problems as myocardial infarction, blood congulation, and carcinogenesis. What can be done to reduce such hazards as "tar" and nicotine, thus leading to the production of a less harmful eigerette, has been discussed.

This afternoon's workshop seems to me to be a variation on the general theme, being in the nature of a movement written in a minor key. It has dealt with certain components in tobacco smoke, e.g., nicotine, whose deleterious properties have not been experimentally and clinically established, but which are nevertheless under various degrees of suspicion. Therefore, these components must be examined in the process of writing the score for the orchestration of Dr. Wynder's symphony, entitled Toward a Less Harmful Cigarette.

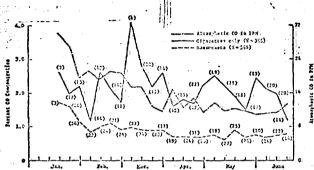
Carbon monoxide (CO) is one of these components of tobacco smoke that has long been suspected of being harmful and, hence, has received much study over the years.

The problem of CO as a harmful constituent of tobacco smoke raises two questions:

1. Does the amount of CO in the blood differ between the smoker audinonsmoker?
2. If more CO is present in the blood of the smoker, does it produce either functional or structural pathological changes? Are such changes demonstrable by symptomatic, clinical, or laboratory evidence, and can they therefore be assumed to be detrimental to the health or well-being of the smoker as is true in the case of other components of tobacco-smoke?

There is abundant evidence in the literature to answer unequivocally the question of the difference between the CO blood level concentration in the smoker and nonsmoker. The article by Larson et al. (1) is replete with references covering studies over the past 50 years of CO blood levels in smokers and nonsmokers under various conditions, as well as the effect of various quantity levels of smoking on the CO blood level.

Numerous studies on the normal blood level of CO in the nonsmoker show ranges from 0.5-2.8%. In our study, we used 1% as the normal level.



Text-result 1.—Distribution median of CO in blood of eigarette-only smokers and nonsmolters.

After analyzing our data, we observed a significant association, which, however, was not noted for every location of the monitoring station. Goldsmith ct. of. (2) who had studied the blood CO levels of long-horemen in San Francisco in relation to their smoking habits suggested that the collected data be used to study the smoking habits of this postmortem population.

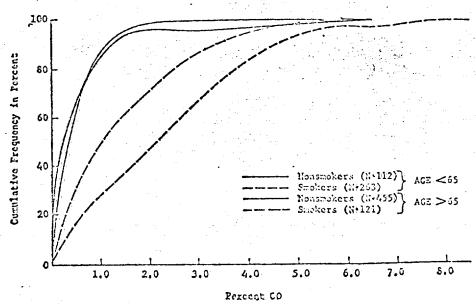
From November 1-June 39, 1991, 2,207 cases were surveyed, and the data were correlated with 1) the CO concentration of the ambient air at certain monitoring stations in Los Angeles and 2) the smoking habits of the study group (3). To determine the smoking habits of the group, a questionneire was mailed to the next of hin, when known, or to a known informant. This reduced the group to a total of 1,578 persons, from whom we received usable smoking histories: for 1,073 persons.

The 1,073 persons were divided into two groups: 1) measurehers (including examples and persons who never smoked), and 2) moders. These two groups were further subdivided into (a) these under age 65 and (b) those over age 65. The blood CO levels in the entire group ranged from 0-11.6%. Over 80% of the nonsmokers, regardless of age, fell in the 1% or less CO level. A blood CO level of 5%, regardless of smoking habit, was considered abnormally high.

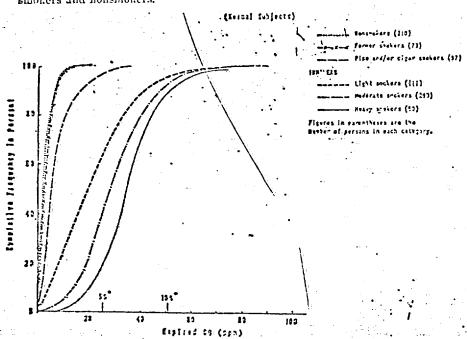
Forty-six persons had values of 5% and all of these were smokers, except 3 who were ex-smokers. Only 7 of the 46 persons were age 65 or over; in other words, 85% of the persons were in the younger age group. Furthermore, with the use of 1% CO as the normal blood level for nonsmokers, 62% of the nonemolters haddless than this level, whereas only 22% of the smokers had values this low (text-fig. 2). Also, the smokers tended to have a much greater frequency at the extreme values of more than 4%. Moreover, smokers over 65 years old had almost twice as high a percentage value under 1% as the smokers under 65 years (text-fig. 2). The interpretation of this finding offers room for speculation, with one possibility being that older smokers might smoke less than their younger counterparts.

Another inferesting fact gleaned from a study of the observed median CO values is that the values of male nonsmokers were greater than those of female nonsmokers by a factor of nearly 2. On the other hand, for smokers, the distribution by sex did not show consistent differences.

That there is a direct correlation between the height of the CO blood level and the number of eigenettes smoked is a well-established fact, as domonstrated by Goldsmith (2) in his study of a group of San Francisco longshoremen (text-fig. 3). This is seen in the graph of percentage cumulative frequency of expired CO measured in ppm as related to the smoking habits of his study group.



Text-riouse 2.—Percentage cumulative frequency and percentage of bleed CO in smokers and nonsmokers.



TEXT-LIGHTS 3.—Distribution of expired CO in long-horemen, by smoking pattern: ILWU study, 1961. Percent carboxylemoglobin concentration based on regression: COIIb% = 0.21 + 0.19 × (CO ppm).

The graphs of Goldsmith's cases of live persons and of our cases show very good correlation incl. a CO expressed in ppm of expired air with that obtained from a study of postmortem blood expressed in percentage terms of carboxyhenoglobin concentration.

Goldsmith did not correlate the CO blood levels with the general health of his subjects; and obviously in our series, we were denied that opportunity, since the deaths we studied included these from natural causes due to disease and also homicidal, suicidal, and recidental deaths. In point of fact neither of these studies answers the question. "Is smoking dangerous to health?"

Fortunately, there is good evidence available which bridges this gap, namely, the study made by Sievers et al. (4) of the effect of exposure to known concentrations of CO on a group of 156 police traffic officers, between 32 and 51 years old, who were assigned to duty in the Holland Tunnel for a period of 13 years. These officers were exposed to an average of 70 ppm of CO, which is equivalent to 10% (COHb saturation), with brief exposures up to 260-360 ppm at times and with the heaviest level for a 24-hour period of 66 ppm (14% COHb). Infrequently, the CO level exceeded 260 ppm (32% COHb) and rarely rose as high as 360 ppm (10% COHb) for a few minutes at a time.

This study on police traffic officers is particularly valuable for the purpose of this workshop, for it demonstrated that these men showed no evidence of injury to their health, as determined by serial physical examinations, blood and urine studies, EKG tracings, blood pressure readings, and neurological examinations. In this latter connection, an excellent test for judging the integrity of the nervous system was the pistol marks—manship record of these officers. The Port Authority pistol team was composed of 7 officers, 6 of whom had tunnel du'y, and the team consistently finished in first or second place in formal mpetition with pistol teams from other police organizations for 7 consecutive years.

Even more pertinent to our charge at this time is the study of the smoking habits of the officers in relation to their blood CO levels. Variation in the entire group ranged from 0.5-12.1% saturation, the highest values being obtained in those who smoked and were stationed on the upgrade section of the tunnel and who were exposed to atmospheric CO readings slightly above 100 ppm (16% COIIb saturation) for a 2-hour period in contrast to the average daily value of 70 ppm (10% COIIb saturation).

What appears to be the most significant observation in this study of traffic officers in the Holland Tunnel is that the blood CO levels of non-smokers in the tunnel on the average exceeded those of smokers in an environment free from any occupational exposure to CO. Since these men remained healthy after being consistently exposed for 13 years to CO levels appreciably higher than those found in tobacco smoke, the conclusion them is inescapable that smokers with CO levels that lie well within these same ranges are similarly unaffected by CO.

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- (5) Cumhin, T. J., Flood, L. P. L., and Peckins, N. M.: Carboxyhemeglobin in relation to all pollution and smoking: Postmortem studies. Arch Environ Health (Chicago) 10: 179-165, 1965.
- (4) Sowers, R. F., Edwards, T. L., Mureay, A. L., and Scholler, H. Hit Effect of exposure to known concentrations of carbon monoxider. Study of traffic officers thation of of the Medical Thornel for T. varis, PANA, 1489 5, 7,589, 1019.

(25) DINMAN B D: Carbon monoxide and cigarette smoking. JAMA 212:

Although the community is concerned with carbon monoxide body burdens arising from ambient concentrations of this gas, it ignores almost totally the most significant source of carbon monoxide intake—the cigarette. This is pradoxical, since the air of our diles rarely contains more than 30 parts of carbon monoxide per milian parts of air (ie, 30 ppm); cigarette smoke streams have been reported to contain from 400 to 40, by ppm carbon monoxide!

The body itself manufactures arbon monoxide in the course of the breakdown of hemoglobin to the extent of about 1 teaspoonful per dy. This small amount of carbon monoxide converts about 0.5% of homoglobin to inactive carboxytemoglobin. By contrast, the light smoker converts about 3% of his homoglobin while the heavy smoker motivates approximately 8% of his blood pigment. Pipe and cigar smokers rarely achieve such loadints. The body has "learned" to slapt to the small amount of selfwoduced carbon monoxide over the ourse of evolution. However, the bdy burden arising from cigarette moking probably extends beyond the limit of ready accommodation.

What is the significance of such garette-caused carbon monoxide tody burdens? It is quite clear that isual acuity at low levels of light Intensity is impaired with carbon monoxide loadings in the middle of hat range of carboxyhemoglobin levels seen among cigarette smokers. Less clear at this time is the effect of carbon monoxide per se upon cardiac function. However, among patients with heart disease whose ability to accommodate is compromised, at levels of 7% to 9% carboxyliemoglobin, there is deterioration in several cardiac-function. indices. On the basis of animal experimental data, it appears that long-term carbon monoxide exposures with about 12% carboxyhemoglobin loading are associated with increased deposition of cholesterol in blood vessels. On the basis

of epidemiological data, there are some suggestions that this might also apply to humans. In addition, research in mountainous areas suggests that the carbon monoxide loading stemming from ciramette smoking contributes significantly to the development of chronic mountain sickness.

- (a) The level of 40,000 ppm carbon monoxide represents pure cigarette smoke which a smoker does not inhale continuously. The concentration of carbon monoxide in the expired air of a heavy smoker is 16.4 ppm and in that of a light smoker 7.7 ppm (see page 19).
- (b) The carbonyhemoglobin levels of 3% as estimated for a light smoker and 8% for a heavy smoker are not supported by the available results of investigations. Balbo et al (1966) reported a mean level of 2.8% for 7 smokers consuming 30 cigarettes daily. Rouch et al (1971) recorded a mean level of 4.25% for 15 smokers using more than 10 cigarettes daily (see page 15).
- (c) The investigation quoted refers probably to Aronow et al (1971), who noted a reduction in exercise tolerance when patients with angina pectoris smoked low-nicotine cigarettes. The carboxyhemoglobin blood level is elevated to 7.79% but a cause-and-effect relationship has not been demonstrated (see page 62).
- (d) The cited work on cholesterol deposition involves rabbits fed with cholesterol in the diet. In dogs, chronically exposed to 100 ppm carbon monoxide, De Bias et al (1972) failed to exaggerate the signs of myocardial ischemia. There is no experimental support for the statement that a blood level of 10% carboxyhemoglobin is harmful to the ischemic heart (see page 62).

(26) DOYLE J T: Smoking and myocardial infarction. Circulation 39 & 40: Suppl 4: 130-43, 1969.

This review contains a paragraph on the role of carbon monoxide in pathogenesis of atherosclerosis. The author views the problem in the proper perspective.

The manner in which eigarette smoking accelerates atheroselerosis and its complications is, in short, unexplained. It is possible that in some way eiggrette smoking damages the arterial intima. Carbon monoxide is the likeliest immediate candidate for such a role. Some presently mysterious interference with the normal mechanism of transport of lipids from the plasma through the vascular tunies to the lymphatics secondary to the inhalation of eigarette smoke is: an alternative possibility: in all populations yet senutinized, the prevalence and incidence of CHID rice with the serum chalesterol concentrationa It is, accordingly, a plausible hypothesis that inordinate eigarette smoking may be associated with an increased serum cholestorol concentration. Such a relationship does, indeed, exist, but is unimpressive. Although the serum cholesterol concentration in both men and women is consistently higher in eigarette smollers, the influence of increasing age is substantially greater (figs., 1 and 2).50 The observation that heavy cigarette smokers have far more atheroma than nonsmokers is, possibly, complemented by Astrup's observation that fat-fed rabbits exposed to high tensions of carbon monoxide exhibit extreme hyperlipidemia and cholesterosis as compared to controls not exposed to carbon monoxide. 3-5, 28 This interesting experimental model has, however, no recognized counterpart in human epidemiological studies. Obesity as a cononary risk factor is not related to eigarette smoking." Dasily, the arterial blood pressure is not associated with eigarette habit.2

FACTS ABOUT SPECIFIC AIR POLLUTANTS

Carlon Monoxide- -

Carbon monoxide is well known to all of us. Yet many smokens are unaware that approximately seven to eight percent of their hemoglobin may be bound as carboxyhemoglobin. If in the mean time, such an individual should develop vascular insufficiency to vital organs, and then be forced to breath ambient air containing 30 ppm of CO for four to six hours or getten into an atmosphere where he would be exposed to 120 ppm CO for one hour, he would bind an additional five percent of his hemoglobin and could suffer tragic results.

The figure of 7% to 8% carbonyhemoglobin among smokers is an overestimation. A review of the literature shows an overall mean of 3.76% for 2,054 smokers 4 to 12 hours after smoking, and a peak level of 5.26% after smoking (see pages 12 to 14 and 20 to 21). (28) GOLDSMITH J R: Carbon monoxide and coronary heart disease. Ann Int. Med 71: 199-201, 1969.

A list of future investigations relating to carbon monoxide is included in this Editorial.

The completed jigsaw puzzle may give a clear picture showing that the role of smoking in atheroselerotic diseases is mediated in part by carbon monoxide through such mechanisms as those in the preceding paragraph. Missing pieces are needed: [1] the effects of carbon monoxide exposure with and without vasoactive agents on production of anginal symptoms with exercise; [2] prognostic importance of carboxyhemoglobin levels over both short and long periods of time in cardiovascular diseases; [3] mechanisms and significance of alteredi hemoglobin binding of oxygen with age, smoking, and other factors; and [4] the possibility of prevention of cardiovascular effects of cigarette smoking when such smoking does not lead to carbon monoxide absorption.

(29) GOLDSMITH J R: Carbon monoxide and coronary heart disease: Compelling evidence in angina pectoris. Ann Int Med 77: 808-10, 1972.

This Editorial is devoted largely to the investigations of Aronow. The 2 paragraphs reproduced herewith contain suggestions for future investigations which have not been discussed elsewhere in this review.

We still lack decisive epidemiologic evidence that there is a risk of more rapid development of coronary heart disease in cigarette smokers with high carbonmonoxide uptake, compared with those of the same age and smoking habits but with low carbon-monoxide uptake. We do, however, have convincing evidence that the death rates from coronary heart disease are higher in cigarette smokers than in nonsmokers (6). The 1972 Surgeon General's smoking and health report says, "Experimental and epidemiological investigations impliente the elevation of carboxyhemoglobin levels in smokers as a contributor to the development of CHD and arteriosclerotic peripheral vascular disease." If one estimates the number of excess deaths caused by arteriosclerotic heart disease in smokers in comparison with deaths of people of the same age and sex who are nonsmokers, the potential for prevention is vast. Of the approximately 580.000 deaths in the U.S. caused by ameriosclerotic heart disease Join Jette smoking makes the greatest proportionate kontibution to the deaths of those under 60 years ok doubling the mortality ratios in several studies. If we can substantially reduce this tell of deathward of associated disability by reducing carbon monoxide exposure, it would be a massive public health achievement. We are challenged to develop an alteration in eigarettesmoking behavior that does not permit an increase in carboxyhemoglobin to occur.

It is conceivable that with a cigarette that has a catalytic filter or in which the combustion processes are altered there would be less uptake of carbon monoxide by the smoker. Possibly an alteration in the type of tobacco used would have such effects. Difference in tobacco type is credited with the differential effect of smoking eigars or pipes. Smoking these produces relatively little increase in carboxyhemoglobin compared with that from cigarettes. Although much attention is given to cigarettes that are low in tar and nicotine, practically no attention has so far been given to the public health importance. of eigarette smoking that produces a low output of carbon monoxide. Such attention is urgently indicated by the evidence that even small increases in carboxyliemoglobin, as we used to think of them. can decrease the work capacity of persons with angina pectoris.

Cigarette Smoking ...

Patients with angina who smoke a single eigentite before exercise experience significant decreases in the duration of exercise required to precipitate ischemic pain.³¹ The decreased exercise capacity after eigentite smoking is associated with a greater heart rate and blood pressure both at rest andiafter equal amounts of exercise. Since the pressure-rate product was the same at angina before and after smoking, Aronow and Kaplan concluded that smoking increased MVO₂ during exercise, thus procipitating ischemic pain sooner without appreciably altering myocardial oxygen delivery. These authors attributed the alteration in circulatory response to exercise to gauglionic stimulation by nicotine, although subsequent studies⁵² using low-nicotine eigerettes yielded the same results as those using ordinary eigerettes. Cigarette smoking might also impair myocardial oxygen delivery in some individuals by converting hemoglobin to carbovyhemoglobin, a change which impairs or destroys the ability of hemoglobin to convey oxygen to tissues. Up to

The last sentence quotes Ayres et al, which is actually an abstract.

The statement that heavy smoking can produce a blood level of 15% carboxyhemoglobin is not based on any experimental observations.

Carboxyhemoglobin (CCMD) and the Access to Oxygent An Example of Human Counter-Evolution

STEPHEN M. AYRES, MD, FACO: STANLEY GIANNELLI, Ir., MD, FACO: HILTRUD MUELLER, MD, New York, New York

Cigarette smoking and exposure to community air pollution produce COHD saturations between 3 and 15% and decrease both oxygen capacity and the unloading tension of circulating hemoglobin. An individual with 15% COHD has regressed to a hemoglobin which is functionally intermediate between that of an elephant and a newborn goat.

Acute studies performed in 26 subjects demonstrated that elevation of COHE levels to an average of 7.98% increased cardiac output from 5.01 to 5.56 liters/min, increased minute ventilation from 6:86 to 8.64 liters/ min, and decreased arterial and mixed venous oxygen tension from 81 and 39 to 76 and 31 mm Hg, respectively. Myocardial studies performed by coronary sinus catheterization demonstrated think similar elevations of COHB increased coronary blood flow, decreased coronary artery-coronary sinus oxygen content and decreased coronary sinus oxygen; tension. The changes were most marked in patients with coronary artery disease or chronic emphysema, lactate extraction decreasing in 10 of 15 patients. The possibility of adaptation was studied by achieving similar COHB concentrations with both a low and high concentration of carbon monoxide. Hemodynamic changes appeared; more marked with administration of the high concentration even though COHE was the same.

These studies suggest that COHB concentrations between 5 and 10% may produce abnormal myocardial function in certain individuals. The well-known deleterious effect of cigarette smoking on the heart may be explained by the interaction of COHB and alcoting the latter increasing cardina work and the former decreasing oxygen availability.

(31) LINDQUIST V A Y: Carbon monoxide: Its relationship to air pollution and cigarette smoking. Public Health London 86: 20-6, 1970.

O Cigars and pipe tobacco produce more CO than eigarettes, but eigarette smokers tend to have higher levels of COUD because they usually inhale. The gas phase of cigarette smoke contains 1-5% CO (Orborne et al. 1965; Politoven & Niessen, 1961) and the concentrations tend to be highest as the eigarette is smoked down toward the butt-end. Continuous exposure to such ambient concentrations would normally render a man unconscious in a few minutes. However, inhalation of degreette smoke is both transient and intermittent and the gas is diluted with atmospheric air so that a single eigarette would not be expected to produce an immediate rise in COHb of more than 3%. During the course of a day, those who smoke heavily and inhale usually have mean COHb concentrations in excess of 4% and may even exceed 10% (Ayres et al., 1966; Goldsmith & Landaw, 1968).

(a) This is a more accurate summary of carboxyhemoglobin levels in cigarette smokers.

Myocardial Oxygenation

Those organs with high oxygen consumption leave little "reserve" in the blood supplying them and therefore rely more on increased perfusion to meet any extra demand for oxygen. The myocardium is a typical example. A combination of high oxygen demand, poor perfusion, lowered oxygen capacity and impaired oxygen uncoupling from the blood will obviously prejudice tissue respiration. This was classically demonstrated by Ayres et al. (1969, 1970). In a group of noncoronary disease patients undergoing cardiac cathete, ration raising the COMb level to 9.6% produced a significant increase in coronary perfusion. Despite this, however, the oxygen tension of coronary sinus blood, and presumably of the myocardium itself, dropped slightly. After raising the COMb to a similar degree in a group with established coronary disease, the increase in perfusion was less marked and there was definite reversal of lactate and pyruvate extraction in addition to a drop in oxygen extraction. This indicated significant myocardial hypexia.

(b) The experiments of Ayres et al (1969, 1970), consisting of inhalation of carbon monoxide causing blood levels of 9.0% are probably in error. This is discussed clsewhere in the present commentary (see page 148).

The next problem is to determine by what mechanism does smoking accelerate thrombosis. Employing the method of multiple working hypotheses, a number of possibilities immediately present themselves. One is that smoking produces an increase in carbon monoxide hemoglobin up to 15-20 per cent which somehow promotes thrombosis. Another is that nicotine absorption itself sets into operation the thrombotic process. A third possibility is the well known effect of nicotine in liberating increased amounts of epinephrine which may be the real culprit in accelerating thrombosis. A fourth possibility is that smoke irritation in the bronchial mucosa liberates into the blood stream a thrombus inducing agent. Perhaps there are other possibilities as yet unidentified

That cigarette smoking produces an increase in carboxyhemoglobin of up to 15% to 20% is a rarity. In a review of the literature, it was found that out of 30 investigations only Meigs (1948) reported a mean level of 16.2% for a group of 6 habitual smokers. The overall mean level for 2,054 subjects reported in 29 investigations was 3.76% (see page 12).

(33) NAHUM L.H.: Tonic products in cigarette smoke: pleasure or poisons Conn Med 32: 154-5, 1966.

Okay, well let's see what it is that the cigarette smoker inhales. Most people when they consider air pollution think of the automobile, the smoke-stack or the trash burners. It's time then to point to a most damaging source of air pollution, the cigarette. One of the toxic products of the automobile exhaust is carbon monoxide (CO). Exposure for one hour to a concentration of this gas of 120 parts per million causes inactivation of about 5 per cent of the body bemoglobin by forming CO hemoglobin. Concentrations of CO as high as 100 ppm. often occur in garages, in tunnels, and behind automobiles. Such concentrations are tiny in comparison with those (12,000 ppm.) found in cigarette smokel

The smoker survives because most of the time he breathes air not so heavily polluted. Nevertheless the smoker can carry 15 to 20 per cent CO hemoglobin for hours and seriously reduce the oxygen supply to already compromised areas in the brain, heart and elswhere whose arteries are narrowed by atherosclerotic disease. Furthermore, in a poorly ventilated smoke-filled room concentrations of CO can easily reach several hundred parts per million thus exposing smokers and non-smokers present to a toxic hazard. The headache and fatigue that those exposed experience after a time in such an atmosphere is no accident and not psychosomatic.

The concentration of carbon monoxide that a smoker is exposed to continuously is not 42,000 ppm. This is the concentration of pure cigarette smoke which reaches the lung diluted with atmospheric air. The concentration of carbon monoxide in expired air would be a more reasonable estimation. Ringold et al (1962) have reported the following observations with regard to the latter: heavy smokers had a concentration of 16.4 ppm, light smokers 7.7 ppm and nonsmokers 0.8 ppm (see page 19).

(34) NAHUM L H: The effects of carbon monoxide on human health. Conn Med 33: 90-2, 1969.

c. CO. occurs in high concentration in eigarette smoke greater than 2 per cent, this means 20,000 ppm, although amestimate of the average concentration in smoke is much less-400 ppm. In a population of longalioremen smoking produced 6 per cent of COHb. When it comes to occupational exposure 12-14 per cent of employed persons had occupations in which there is a likelihood of exposure. Vanious forms of indoor combustion may emit CO; and a number of deaths each year are due to poisoning from this source. Gas fired base-board heaters were incriminated by Michigan State Department of Public Health. Open fires and charcoal braziers produce substantial amounts of CO.

(a) Blood levels of habitual smokers have been reported by 11 groups of investigators. The overall peak after smoking was 5.26% (see page 20). The blood levels 4 to 12 hours after smoking have been reported by 30 groups of investigators with an overall mean of 3.76%. The smoker throughout 24 hours sustains a level between 3.76% and 5.26% (see page 12).

V Grut² alleged that 46 per cent of 721 drivers had chronic CO: poisoning characterized by fatigue, headache, irritability, dizziness, disturbed sleep and other symptoms. Some subjects had abnormal neurological symptoms. From the epidemiological point of view it is desirable to obtain data which would show whether there are CO: associated increases in such relatively frequent events as motor vehicle accidents or in fatality rates with myocardial infarction to confirm the data from the Los Angeles Hospitals where an association of CO, and case fatality rates in 3,080 patients with myocardial infarction was observed. The central nervous system effects are definitely due to anoxia. The mechanism of myocardial effects probably are similarly produced. Lindenberg³ did obtain significant electrocardiognaphic changes on exposing dogs for six weeks to 50 ppm. CO. They also showed dilatation of the right ventricle, scarning of heart muscle and fatty degeneration. A very important question for epidemiologists to study is whether exposure to low concentrations of CO: have a role in the development of human heart disease. Inferring from results. of acute toxicologic and experimental studies, we can begin to appreciate the abundant data linking cigarette smoking to coronary heart disease. As far as eigarette smoking is concerned we must keep in mind that high levels of COIIb, imply also increased respiratory absorption of other ingredients of tohacco smoke.

It is safe to say that exposure to CO, is wide-spread, that the smoker who inhales 6 per cent CO, is developing a blood concentration which is a serious threat to the health in persons with underlying cardiovascular disease. It is also true that community air pollution may produce COHb, in non-smokers similar to that observed in smokers. Even low and commonly occurring CO, exposures may impair performance of complex psychomotor tasks. Finally that CO, has a tole in motor vehicle accidents, is supported by data of high COHb, in many drivers involved in accidents.

L.H.N.

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- 2. Grut, A.: Chronic caroon monoxide poisoning, (Munksgaard) Copenhagen, 1940, p. 44.
- 5. Lindenberg, R., Levy, D., Preziosi, T. and Christenson. M.: paper presented at a meeting of the American Industrial Hygiene Association, Washington, D.C., 1992.
- 4. Nahum, L. H.: Toxic products in rigarette smoker pleasure or poison. Conn. Med., 32: 154, March, 1968.
- (b) Each statement in this paragraph can be challenged by work of other investigators. For instance, at a meeting Lindenberg et all presented the electrocardiographic changes on exposing dogs for 6 weeks to 50 ppm carbon monexide. De Bias et al (1972) published the results of their investigations indicating that exposure of dogs to 100 ppm carbon monoxide for 14 weeks does not influence the heart that has been previously infarcted (see page 62).

- (35) ROSE E F and ROSE M: Carbon monoxide: A challenge to the physician. Clin Med. 78: 12-21, 1971.
 - as The amount of CO in eighrence smoke varies between 1 and 2.5 per cent by volumed and can create discomfast not only for the addict but also for others in the environment. For the average smolter, the concentration of CO reaching the alveoli is about 0.04 per cent (400 ppm). The importance of cigarette-produced CO is amply demonstrated by samplings from fleet submarines submerged for extended periods. In: 30 hours, concentrations may reach 0.01 per cent (100 ppm), with cigarette snoke accounting for 75 to 80 per cent of the CO production. This exceeds the allowable maximum concentrations recommended by the American Conference of Governmental Industrial Mygienists, which states that CO concentrations in the atmosphere should be kept below 50 ppm.7
- (a) The concentration of carbon monoxide reaching the alveoli is not 400 ppm for the average smoker. Jongbloed (1939) noted the highest level of 31.5 ppm after a subject finished smoking a 4th cigarette (see page 19).
 - Because individuals who smoke from 20 to 30 cigarettes daily have a COITb level ranging from three to 10 per cent,16 there is a widespread mistaken idea that smokers are more susceptible to environmental CO than are nonsmokers. CO from cigarettes and CO in the ambient air are not additive in their biologic effects. CO is absorbed into the bloodstream only when the pressure of CO in the ambient air exceeds that in the pulmonary capillary blood. Thus, persons with COHb levels of five per cent as a result of smoking do not absorb further CO unless the environmental CO concentrations exceed 0.003 to 0.004 per cent.

(b) The cited reference 16 is Barach et al (1941). Although the range is 3% to 10%, the mean value is 5.7% for the carboxyhemoglobin level. Other groups of investigators reported mean values of 4.3 and 4.85% (see page 21).

When cardiac function is novmal there is a significant margin of safety even though the CO intoxication is of long duration. A variety of electrocardiographic aberrations have been observed following CO poisoning, but typically there is a low-voltage pattern.73 Recovery is apparently rapid following the restoration of oxygen and there is a reversion to a "normal" electrocardiogram; however, enzyme studies may show alterations indicating ischemic heart damage.22 Epidemiologic studies of eigarette smokers indicate that the death rate from coronary heart disease is considerably higher for smokers than for nonsmokers,24 and in patients with heart disease there is deterionation of cardiac-function indices at blood levels of seven to nine per cent COHb 35

(c) The cited reference for the last sentence is by Dinman (1970), an editorial that is commented upon elsewhere in this report (see page 160).

(36) SELTZER C C: The effect of cigarette smoking on coronary heart disease. Arch Environ Health 20: 418-23, 1970.

This article reviews the effect of cigarette smoking on coronary heart disease. Soltzer discusses the shortcomings of interpretations appearing in the Surgeon General's report. The question as to the role of carbon monoxide in cigarette smoke is discussed. It should be noted that the work of Ayres quoted in the article has been commented upon elsewhere (see page 148).

6. Does the carbon monoxide constituent of cigarette smoke result in or contribute to increased myocardial infarction or sudden death either in normal individuals or in persons with already impaired coronary circulation due to CHD?

Studies have shown that the carbon monoxide constituent of eigerette smolie does effect increases (2% to 10%) in the levels of carboxylamoglobin (COIb) saturation when heavy eigerette smokers and measmokers were compared, with the consequent displacement of explamoglobin. In addition, earbon monoxide effects a shift to the left of the exygen-hemoglobin dissociation curve, which may result in a decreased release of exygen at the tissue-level.²³

On the whole, experimental and clinical investigations bearing on this question are few. The most calient work in this area has been performed by Ayres and associates. In 23 human subjects before and after carbon monoxide inhalation, these investigators found no significant change in oxygen tension. In another experiment, after exposure to carbon monoxide, coronary blood flow increased significantly in seven non-CHD patients but not lin four patients with arteriographically proven ChiD. In the patients with ChiD, myocardial lactate and pyravate extraction decreased or shifted to actual production, suggesting annearobic metabolism.

If carbon monoxide does in fact appreciably decrease oxygen extraction at the myecordial level, the matter of oxygen consumption may hinge on the extent of increase in coronary blood flow in normal persom, while in persons with diseased coror'ry arteries, the increase in blood flow is slight or absent, Hence, it may be a question of the ultimate balance of these opposing forces. In normal persons, there is the presumption that the increased coronary blood flow more than matches the presumed decrease in oxygen extraction. Whether or not this fails to occur in patients with obvious CHD, to such an extent as to "trigger" a coronary event is as yet unknown and much work remains to be done in this area.

X. RECOMMENDATIONS

Three types of investigations are recommended to supplement the available information on the role of carbon monoxide in eigarette smoking.

They are as follows:

- smokers. Most of the reported investigations pertain to the blood level before and the peak level after cigarette smoking. It is necessary to take hourly samples in the course of a day and less frequently samples at night, to derive an integrated blood level. In the same investigation, the subjects, both smokers and nonsmokers, will be exposed to varying levels of muscular activity and will include some with chronic pulmonary or coronary heart disease.
- 2. Comparison of cigarette smoking and carbon monoxide inhalation.

 Of all the investigations on the effects of inhalation of carbon monoxide in man and animals almost none extend to a comparison of the equivalent carboxyhemoglobin level with that produced by cigarette smoking. Investigators should be encouraged to include cigarette smoking in their investigation of carbon monoxide inhalation.

 Special procedures that will determine the development of tolerance to carbon monoxide and the interaction between carbon monoxide and other constituents of cigarette smoke may be added to the investigation.

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- 3. Retrospective studies of workers in an environment containing carbon monoxide. Tunnel workers and miners may be conveniently studied for incidence of coronary heart disease. The limited number of reports on such individuals so far fail to show any disturbance that could be attributed to chronic exposure to carbon monoxide.

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ANTONINI, CHIANCONE and BRUNORI 1967 (A64)180
ANTONINI, SCHUSTER, BRUNORI and WYMAN 1965 (A 61) 180 ANTOS and SEVCIK 1971 (393) 76, 74 AOYAMA 1970 (A 272) 194 APPLEBY 1969 (A 66):180 AQUINAS 1964 (A 729): 223 ARAI 1969 (A 466) 206 ARIN and WARNECK 1972 (A 151) 186 ARMSTRONG 1922 (109) 30, 29 ARNOTT, PETIT and CHABRIER 1964 (A 467) 206 ARONDEL, GAUBERTI and ROCHET 1964 (A 650) 218 ARONOW, DENDINGER and ROKAW 1971 (311): 64, 63; (647) 114, 147 ARONOW, HARRIS, ISBELLL, ROKAW and IMPARATO 1972 (312) 64, 62; (649) 114, 147 ARONOW, KAPLAN and JACOB 1938 (646) 114, 147 ARONOW and ROKAW 1971 (313) 64, 64; (648) 114, 147 ARTURSON, GARBY, ROBERT and ZAAR 1972 (35) 17, 15; (142) 34, 34 ASAI and TORU 1969 (A 468) 206 ASODRBE DOMINGUEZ 1969 (A 790) 229. ASMUSSEN and VINTHER-PAULSEN 1953 (272) 59, 57 ASMUSSEN and VINTHER-PAULSEN 1949 (394) 76, 75 ASTRUP 1964 (360) 72, 69 ASTRUP 1966A (361) 72, 69 ASTRUP 1966B (362): 72, 69 ASTRUP 1967 (363) 72, 70 ASTRUP 1969 (304) 72, 70 ASTRUP' 1970 (A: 67): 180: ASTRUP 1970 (365) 72, 70 ASTRUP 1972 (366): 72, 70 ASTRUP 1966 (367) 72, 66 ASTRUP 1967 (368) 70, 70 ASTRUP, KJELDSEN and WANSTRUP 1970A (369) 72, 70 ASTRUP, KJELDSEN and WANSTRUP 1970B (370) 72, 70

ASTRUP, KJELDSEN and SIGAARD-ANDERSEN 1971 (479) 91, 89 ASTRUP, PAULI, KJELDSEN and PETERSEN 1968 (200) 46, 45 ASTRUP, TROLLIE, OLSEN and KJELDSEN 1972 (563) 105, 103, 104 AUBEAU, LEROY and CHAMPEIX 1965 (A 320) 197 AYRES 1972 (652) 114, 149 AYRES and BUEHLER: 1970 (169): 40, 38 AYRES, BUEHLER and ARMSTRONG 1964 (A 36) 200 AYRES, CRISCITIELLO and GIANNELLI 1956 (A 4) 176 AYRES, EVANS and BUCHLER 1972 (A 245) 192 AYRES, GIANNELI and ARMSTRONG 1965 (A 36):17, 15; (143) 34, 33; (193) 44, 42 AYRES, GIANNELLI and MUELLER, 1970 A314) 64, 63; (650) 114, 148 AYRES, MUELLER, GREGORY, GIANNELLI and PENNY 1969 (315):64, 63; (651) 114, 148 BACK 1969 (499): 94, 93 BADAL 1964 (A 469) 206 BADEN 1970-(616) 112, 111 BAKER and TUMASONIS 1971 (A 582) BAKER and TUMASONIS 1972 (A 583) 214 BAKER, FISHER, MASEMORE and SOPHER je. 1972 (A: 273): 194 BALBO, MARUCCI and RONCHI 1966 (37) 17, 13, 15; (144) 34, 33 BANERJEE, DOUZOU and LOMBARD 1968 (A 68) 180 BANKL and JELLINGER 1967 (564) 105, 102 BANYAI 1970 (653): 115, 151 BARACH, ECKMAN and MOLOMUT 1941 (66) 22, 20, 21 BARBOSA and PETERS 1971 (A 584) 214 BARIBAUD, YACOUB, FAURE, MALINAS and CAU 1970 (565) 105, 103 BARRETT, BENNETT and BUCKMASTER (A 321) 197 BARRIOS, KOLL and MALORN 1969 (500) 94, 93 BINNENBRUCK, HAUSEN, RUNOW and BARTEK, GAUME and ROSTAMI 1970 (A 274) 194 BARTH, 1970 (A 275): 194 BARTHE, PARIS, DUCHEMIN and THOMAS 1953 (145) 34, 33; (38) 17, 14 BARTHELEMY, PARC, MICHAUD and MATHE 1967 (A 791) 229 BARTLETT 1968 (654) 115, 153 BASMADZHIEVA, KURCHATOWA, DAVYDKOVA and TSWETANOV 1968 (A 688) 222 BATES, CHRISTIE and VARVIS 1960 (A 362) 200 BAUER 1965 (A.5) 176 BAUMBERGER: 1923 (110) 30, 29. BAXTER and HOBBS 1967 (111): 30, 28 BEAN 1968 (A 620) 216 BEARD 1969: (1) 9, 6

BEARD and GRANDSTAFF 1970 (480) 91, 88

BEARD and WERTHEIM 1967 (501) 94, 93

BEARON 1965 (A 470) 206 BEAUDOING, GACHON, BUTIN and BOST 1969 (566) 105, 102 BECK and SUTER 1938 (316), 64, 63 BEDELL and OSTIGUY 1967 (A 363) 200 BEECKMANS 1967 (A 6) 176 EEEREL and VANCE 1965 (A 364) 200 BEGHE' 1964 (A 730) 225 BEHRMAN, FISHER and PATON 1971 (567) 105 BELAISCH 1954 (A 731): 225 BELLI and GUILIANO 1955 (67) 22, 20 BELYAEV 1967 (a 546) 211 BENDER, GOTHERT, MALORNEY and SEBBESSE 1971 (481): 91, 89 BENESCH, GIBSON and BENESCH 1964 (A 69) 180 BENESCH, MAEDAN and BENESCH 1972 (A 70): 180 BENSON and GREENBERG 1969 (438) 83, 81 BEREZIN and PUZARES 1969 (A 792) 229. BERNHARD and FILLER: 1968 (A 793) 229 BERTIN, FRANCOIS, PEQUIGNOT and SOULAIRAC 1970 (A 651) 218: BERTONE 1965 (A 471) 206 BESZNYAK 1967 (A 794) 229 BETHEUIL and DELAHAYE-PLOUVIER 1967 (A 652): 218: BETHLENFALVAY 1971a: (395): 76, 74 BETHLENFALVAY 1971B (396) 76, 74 BETKE 1968 (A 72) 181 BETKE and SHEPARD: 1968: (A 71) 180 BEUMER 1964 (A. 365): 200 BEUMER 1965 (A 366) 200 BHATNAGAR 1970 (A 547) 211 BHOWN 1969 (39): 17, 15 BIDE and COLLIER 1964 (A 73): 181 BILCHIK, MULLER-BERGH and FRESHMAN 1971 (439) 83, 81 BILS and ROMANOVSKY 1967 (207) 49, 48 BIMONTE, PORTOLANO and TUFANO 1971 (A 795): 229 BINET and BURSTEIN 1948 (333) 67, 66 BINET and BETOURNE 1951: (273) 59, 57 WERHEIT 19.70 (A 591) 214 BIRNSTINGL, COLE and HAWKINS 1966 (371) 72, 69 BIRNSTINGL, COLE and HAWKINS 1967 (398) 76, 75 BIRNSINGL, BRINSON and CHAKRABARTI 1971 (397) 76, 74 BJURE 1965 (A 367) 200 BJURE and FALLSTROM 1963 (568): 105 BJURE and NILSSON: 1965 (A 7): 176 BLACKMORE 1970: (399) 76 BLACKMORE: 19.70: (A 8) 176 BLACKMORE 1970 (A 165) 187 BLANC, HUYNH and ESPAGNO 1967 (A 322) 197 BLOOM: 1972 (A 166) 187 BLUMER 1970 (A 276) 194

ALLUIDNI bes CHOCADOOR 1968 (A 277) 194 BOCCALETTI, NOFRINI, JOSI and MAGGIO 1966 (A 796) 229 BOECK 1958 (170) 40, 38 BOGDAN and JUCHAU 1970 (A 548): 211 BOKHOVEN and NIESSEN 1961 (112): 30, 28 BOLLINELLI, ROUCH, PUJOL, CARRIERE and CARLES: 1971 (A 368) 200 BONNET, GRATADOU, BONNET and LECIAK 1967 (A 472) 206 BOOZ 1969 (A 369) 200 BORBELY 1965 (A 732) 225 BORST 1967 (275) 59, 57 BOTTEAU and MOUSSION 1957 (A 323) 197. BOUHUYS, GEORG, JONSSON, LUNDIN and LINDELL 1960: (A 370) 200 BOULETREAU and MOTIN: 1970: (A 733) 225 BOULEY, GODIN, ROUSSEL and GIRARD 1971 (A 585): 214 BOUR 1964 (A 654) 218 BOUR, GUY-GRAND, TUTIN and TAMINIAUX 1967 (A 473) 206 BOUR, GUY-GRAND, ROGER, TUTIN and DORF 1968 (599), 110, 108 BOUR, PASQUIER and BERTRAND-HARDY 1966 (A 734) 225 BOUR, TUTIN and PASQUIER 1967 (A 474) 206 BOVE and SIEBENBERG 1970 (A 278): 194 BOWDEN and WOODHALL 1964 (68) 22, 21; BOZEK, PAJOR and WASOWICZ 1965 (A 653) 218 BRAJA and TROMPEO: 1964 (201) 46, 45 BRANDENBERGER 1967 (A 621) 216 BRANDON 1970 (A 735) 225 BRANDT 1965 (A 279) 194 BRAUSER, VERSMOLD and BUCHER 1968 (A 549) BREITNECKER 1938 (429) 80, 79 BRETON, CAROFF, MARTIN, DEHOUVE and DEHOUVE 1969 (430): 80, 79 BRETON, GARAT and DEROBERT 1969 (A -34) BREU 1942 (230) 53, 51 BREU 1942 (274) 59, 57 BREU 1943 (231) 53, 51 BREWER, EATON, GROVER and WEIL 1971 (41) 17, 15 BREWER, EATON, WEIL and GROVER 1970 (40): 17, 15 BREWER 1937. (334): 67, 66 BREYSSE, BOVEF and GABAY 1966 (A 9) 176 BREYSSE and BOVEE 1969 (171) 33, 39 BRICE and ROESLER 1966 (A 280) 194 BRIDGE and CORN 1972 (113) 30, 28 BRIGATTI, PARIGI and VARETTO 1964 (A 689) 222 BROBERG and SMITH 1967 (A 586) 214 BRODY and COBURN 1969 (276) 59, 56; (400) 76, 71 BRODY and COBURN 1970 (277) 59, 56

BRUNNER 1939 (232): 53, 52

BRUNORI 1966 (A 74) 181 BRUNORI, ANTONINI, WYMAN, TENTORI VIVALDI and CARTA 1968 (A 75) 181 BRUNORI, BONAVENTURA, BONAVENTURA and W.YMAN 1972 (A 76) 181: BRUSADELLI, PALMA and DiPRETORO 1968 (A 797) 229 BUCHWALD 1969 (172) 40, 38, 39 BUCHWALD 1969 (A 10): 176 BUCKLEY and FEAR 1964 (A 690): 222 BULTERIJS 1965 (A798) 229 BUNCHER 1969: (569) 105, 103 BURCK and PORTWICH 1964 (551) 101, 99 BURG and DOUGLASS 1969 (A 655) 218 BURGESS, GILLESPIE, GRAF and NADEL 1968 (A 371) 200 BURMEISTER, BARCKOW, HUMPERT, IBE and LERCHE 1968 (A 736) 225 BURMEISTER and HEUHAS 1970 (A 737) 225 BURSTON: 1969. (A 622) 216; (A 799); 229. BURVILL 1970 (623): 216 BYSTROM 19.70: (281): 194 CACCURI 1955 (278) CAILAR, SERRE, ROQUEFFEUIL, LEFEBVRE and MA:LZAC 1968: (A 800): 229 CAIRNS and DENHARDT 1968 (A 587) 214 CALLIGARI 1968: (A 656): 218 CAMMA 1967 (655): 115, 154 CAMPBELL 1936 (208) 49, 48 CAMPBELL 1968 (A282) 194 CANDURA and CRAVERIA 1964 (401) 76, 74 CANEPA, CAVALLO and MUZIO 1968 (A 372) 200 CAPELLARO 1964 (A 691) 222 CAPELLARO and BRAGUZZI 1964 (A 692) 222 CAPELLARO and GANDOLFO 1964 (233) 53, 52 CARDING 1968 (502) 94, 93 CARNOW 1971 (A 246) 192 CAROFF, DEHOUVE and DEROBERT 1970 (279) 59, 57 CARUSO and BARNABA 1968 (A 738) 225 CASARETT 1971 (2) 9 CASCINI and GAZZERRO 1966 (A 693) 222 CASTELLINO 1955 (69) 22, 20. CASULA, NISSARDI, SANNA-RANDACCIO and FRAU 1969: (373): 200 CASULA, NISSARDI, SANNA-RANDACCIO and FRAU 1969 (A 374) 200 **Q**AUGHEY: 19.70: (A: 78): 181: CAUGHEY, ALBEN, McCOY, BOYER, CHARACHE and HATHAWAY 1969 (A 77) 181 CELEGIN, HANSSON and SUNDSTROM 1971 (A: 324): 197-CENTI and ZAFFIRI 1971 (A 73(225 CHANCE, ERECINSKA and WAGNER 1970 (A 550) 211 CHEAH 1970 (A 79) 181 CHERKAVSKIR 1970 (A 657) 218 CHEVALIER, KRUMHOLZ and ROSS 1963



(280) 59, 56

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CHEVALUER, KRUMHOLZ and ROSS 1963 (280): 59, 56 CHEVALIER, KRUMHOLZ and ROSS 1966 (191) 44, 43 CHEW, HANSON and STLACK 1969 (801): 229 CHICHET-GOSSET, CHASSON, OHRESSER, ARNAUD and DUBOULOZ 1967 (A 802) 229 CHICHIKALDO, BENEVELSKY and MINSKY 1966 (A 263) 194 CHINET, MICHELI and HAAB 1971 A 375) 201 CHIODI, DILL, CONSOLAZIO and HORVATH 1941 (195) 44, 42 CHOSY, GEE and RANKIN 1963 (196) 44, 43 CHOVIN 1967 (173) 40, 38, 39 CHRISTIANSEN and MAGID 1970 (A 80). 181 CHUDZINIEWICZ 1957 (335) 67, 66 CHUKHRIENKO and LULKO 1968 A 740): 225 CINKOTAL and THOMSON 1966 (A: 376) 201 CIOCATTO and PATTIONO 1964 (A 741) 225 CIOCATTO, PATTONO, QUERCI and ZAFFIRI 1966 (A 803): 229 CIS and PERANI 1964 (458) 86, 85 CIUHANDU, DIACONOVICI, KISS and RUSU 1964 (325) 197 CIUHANDU, DIACONOVICI, KISS and RUSU 1968 (377) 201 CIUHANDU and RUSU 1968 (A 326): 197-CIUHANDU, RUSU; DIACONOVICI and KISS 1966 (A 11) 176 CLARK and BUCKINGHAM 1971 (A 327) 197 CLAUZEL, TRINQUET, CARRE and MEYER 1966 (197) 44, 43 CLAYTON 1969 (A 247) 192 COBURN 1967 (A 206) 190 COBURN: 1970 (3) 9, 7= (A:154) 186 COBURN: 197.01 (A. 208): 1901 COBURN 1970 (A 209) 190 COBURN, FORSTER and KANE 1965 (A 152) 186 COBURN and MAYERS 1971 (617) 112 COBURN, SWERDLOW, LUGMANMAKI, FORSTER and POWELL 1968 (A 153) 186 COBURN, WALLACE and ABBOUD 1971 (210) 190 COBURN, WILLLAMS and KAHN 1966 (A 205) 190 COBURN, WILLI AMS, WHITE and KAHN 1967 (A 207) 190 COHEN, DEANE and GOLDSMITH 1969 (317) 64, 61 COHEN, DORION, GOLDSMITH and PERMUTT 1971 (174): 40, 38, 39 COHEN; PERKINS, URY and GOLDSMITH 1971 (70) 22, 19 COLE, HAWKINS and ROBERTS 1972 (570) 105, 104 COLLINS and GOULDING 1971 (A 742) 225 COLLISON, RODKEY and O'NEAL 1968

COLTMAN and DUDLEY 1969 (A 212) 190

COLTMAN, DUDLEY and LEVERETT 1969 (211) 190 COMMINS and LAWTHER 1965 (A 13) COMSTOCK, SHAH, MAYER and ABBEY 1971 (571) 105, 102 CONKLE, MABSON, ADAMS, ZEFT and WELCH 1967 (A 167) 187 CONNEY, LEVIN, IKEDA and KUNTZMAN 1968 (A 551) 211 CONROY 1969 (656) 115, 155 COOPER 1966 (4): 9, 8 COOPER, SCHLEYER and ROSENTHAL 1970 (A 552) 211 CORET and HUGHES 1964 (336) 67, 66 COSBY and BERGERON: 1963 (234): 53, COSCIA, PERRELLI, GAIDO and CAPELLARO: 1964 (402): 76, 74 COSIC, ARSENIJEVIC, KANDIC and GRBESA 1964 (A 694) COTES, DABBS, EVANS and HOLLAND 1972 (A 378) 201 CRAMOND 1968 (A 475) 206 CROSETTI, PETTINATI and RUBINO 1965 (A 658) 218 CROSETTI, RUBINO and PETTINATI 1966 (71) 22, 20; (A 81) 181 CUCHE and BERNARD 1969 (A 659) 218 CULVERWELL 1915 (114) 30, 27 CURPHEY, HOOD and PERKINS 1965 (42) 17, 13, 15; (147) 34, 33 CURPHEY 1968 (657) 115, 156 DAHMSTROM, NORDSTROM-OHRBERG and ROTHSCHILD 1958 (43) 17, 14; (148) 34,33 DALGAARD 1965 (A 660) 218 DALHAMN, EDFORD and RYLANDER 1968 (11t) 30, 28 DALLE, TOURNAIRE. BRUDIEUX and DELOST 1971 (A 381) 201 DALY 1969 (A 380) 201 DALY and WALDHAUSEN 1967 (A 379) 201 DAMIA, SIBILLA and DAMIA 1965 (A 743) 225 D'AMORE, GIORDANO and PENATI 1968 (A 476): 206 DANTO: 1964 (618): 112, 111 D'ARCA, GUALDI and ARCIERI 1964 (202) 46, 45 DATSENKO 1964 (A 588) 214 DATSENKO 1965 (A 14) 176 DATSENKO 1965 (A 695) 222 DATSENKO 1966 (235): 53, 52 DATSENKO, DOTSENKO, MARTYNIUK and PAILCHEVSKY 1965 (A 553) 211 DATSENKO, DOTSENKO 1967 (A 554): 211 DAVID 1971 (624) 216 DAVIES, JONES and WARNER 1965 (A 328) 197 DEANE, GOLDSMITH and TUMA 1965 (A 696)

DeBIAS, BIRKHEAD, BANERJEE, KAZAL, HOLBURN, GREENE, HARPER, ROSENFELD MENDUKE, WILLIAMS and FRIEDMAN 1972 (318) 64, 62 DeBRUIN 1967 (175) 40, 38 De BRUIN and HAERINGEN 1965 (176) 40, 38, 39 DeBRUIN, VROEGE and VAN HAERINGEN 1965 (177): 40, 38 DEEN 1969 (804): 230 DeGRAFF, TAYLOR, ORD, CHUANG and JOHNSON 1965 (A 382) 201 DELIVORIA-PAPADOPOULOS and COBURN 1972 (572) 105, 102 DELWICHE 1970 (A 188) 189 DEMANGE and AUZAS 1969 (482) 91, 90 DESBAUMES 1968 (A 698) 222: DESOILLE 1967 (178) 40, 38 DESOILLE, CASTILLON, du PERRON, CREMER and LEBBE 1963 (A 213): 190 DESOILLE, CREMER and GIRARD 1965 (A 214) 190 DETTORI and SCANSETTI 1965 (A 699): 222 DeVALOIS and SCHADE 1967 (529) 97, 96 DEVIATKA 1956 (337): 67, 66 DIAMANT-BERGER, GAJDOS, RAPIN and GOULON 1970: (281): 59, 56 DILLE and MOHLER 1969 (A 168) 187 DIMIZIC, FELICI and BIETTI 1969 (A.477) 206; (A 661) 218 DINMAN 1968 (5) 9, 7 DINMAN 1969 (319) 64, 61 DINMAN 1970: (658) 115, 160 DINMAN, EATON and BREWER 1970 (A 82) DIXON 1927a (116) 30, 27 DIXON 1927b (117) 30, 27 DOBOSZ and LUCZYWEK 1971 (A 478) 206 DOMINGUEZ, HALSTEAD and DOMANSKI 1964: (A 15) 176 DONATELLI 1940 (236) 53, 52 DONTENWILL 1967 (210) 49, 48 DONTENWILL 1970 (211) 49, 48 DONTENWILL, RECKZEH and STADLER 1966 (72) 22, 20; (209) 49, 48 DONTENWILL, RECKZEH and STADLER 1967 (73) 22, 20 DORSCH and KOSTER 1965 (a 16) 176 DOUGLAS 1967 (A 189) 189 DOUMER and MERLEN 1946 (237) 53, 52 DOUZE 1971 (A 662): 218: DOUZE, HEYST, KREUKNIET, LEEUW and HAMELINK 1967 (A 663) 219 DOYLE 1969 (659) 115, 162 DRABKIN 1970 (A 215) 190 DRISCOLL, DEUBER, BAETTIG and GRANDJEAN 1972 (74) 22, 20 DROGICHINA and RYZHKOVA 1967. (A 479) DUBLIN 1972 (94) 26, 25 DuBOIS 1970 (6) 9, 7 DUBOIS and MONKMAN 1972 (A 330): 197

DUBOIS, ZDROJEWSKI and MONKMAN 1966 (A 329) 197 DuCAILAR, LEFEBVRE, ROQUEFEUIL, MALZAC and PROST 1970 (A 805) 230 DUCROS 1968 (A 331) 197 DUGNAT 1965 (503) 94, 93 DUKE, GREEN and NEIL 1952 (212) 49, 47 DUKE and KILLICK 1952 (213): 49, 47 DUNLAP 1961 (A 284) 194 DUPLAY, ZIEGLER, GARDO and PINTO 1967 (403) 76, 74 DVORAK, PROKSAN and ZITKA 1951 (238) 53, 52 EBERSOLD 1958 (A 169) 187 ECKARDT, MacFARLAND, ALARIE and BUSEY 1972 (214) 49, 48 EFFENBERGER 1967 (A 17) 176 EHRICH, BELLET and LEWEY 1944 (239) EHRISMANN and ABEL 1934 (118) 30, 29 EISEN and HAMMOND: 1956 (404): 76, 75 EISENBUD and EHRLICH 1972 (A 248) 192 EL-ATTAR 1968A (405) 76, 74 EL-ATTAR 1968B (406) 76, 74 EL-EBRASHY, EL-ASHMAWY and ALY 1967 (407) 76, 74 ELFIMOVA and KHUCHATURYAN 1968 (A 249): 192 ELIOT and BRA TT 1969 (408) 77, 75 ELLIS and SEATONBERRY 1966 (A 170) 187 ENGEL, RODKEY, O'NEAL and COLLISON 1969 (A 83) 181 ENGEL, RODKEY and KRILL 1971 (A 18) 177 ENVIRONMENTAL HEALTH SERVICE 1970 (7) 9. 7 ENVIRONMENTAL PROTECTION AGENCY 1971 (A 250) 192 EPSTEIN 1969 (A 251): 192 ERBEN 1967 (A 19) 177 ESTABROOK, FRANKLIN and HILDEBRANDT 1970 (A 555) 211 ESTLER 1965 (A 744) 226 ESTLER, AMMON and HEIM 1971A (605) 94. 93 ESTLER, AMMON and ZIMMERMANN 1969 (504) 94, 93 ESTLER, HEIM, AMMON and ZIMMERMANN 1971B (506) 94, 93 FABRE, TRUHAUT and BERROD 1951 (75) 22, 21 FAIVRE, DUREAX, VINCENT and MULLER: 1954 (240): 53, 52 FAIVRE, GILGENKRANTZ and HUEBER 1959 (241) 53, 52 FALCONER and MOLLER 1971 (A 20) 177 FALLSTROM 1968 (A 218): 190: FALLSTROM 1968 (A 219) 190 FALLSTROM 1969 (A 220): 190: FALLSTROM and BJURE 1967 (A 216) 190 FALLSTROM and BJURE 1968. (A 217) 190-FARBEROW and SIMON 1969: (A 625) 216

FATINI and GALLENGA 1968 (A 626): 216-

FAIRE, VINCENT, ESCHAPASSE, CASTAING, LOISEAU and CHEVAIS 1965 (530) 97, 96 FAURE, VINCENT, ESCHAPASSE, LOISEAU and CASTAING 1965 (A 480) 207 FAZEKAS 1967 (507) 94 FELDMAN and LAMPERT 1968 (A 664) 219 FELDSTEIN 1965 (A 21) 177 FELDSTEIN 1965 (A 332) 197 FELDSTEIN: 1967 (A333): 198: FELDSTEIN 1969 (A 252) 192 FENN:1970 (A 155) 186 FENN 1971 (A22) 177 FIANDACA and VERCELLOTTI 1964 (17.9) 40, 37 FINCK 1966 (8) 9, 6 FIRST and MURPHY 1970 (A 700) 222 FISHER, HYDE, BAUE, REIF and KELLY 1969 (198) 44, 42; (215) 49, 48 FISHER 1968 (A 665) 219 FLAXMAN: 1939: (282) 59, 56 FLEIG 1908 (A 589) 214 FLETCHER 1972 (A 383) 201 FODOR 1969 (431) 80, 79 FODOR, MALORNY and COLMANT 1964 (531) 97, 96 FORBES 1970 (A 385) 201 FORBES, SARGENT and ROUGHTON 1945 (A 384) 201 FORMAN and FEIGELSON 1971 (A 84) 181 FORSTER 1970 (A 156): 186 FORSTER, ROUGHTON, CANDER, BRISCOE and KREUZER 1957 (A 386) 201 FORTUNATO and CATALANO 1970 (459) 86, 85 FOURNIER 1967 (A 745): 226 FRANCHINI, CANALE and CELESTRI 1967 (A 23) 177 FRANCISCO and SILVEY 1971 (A 590)214 FRANCOIS and BERTIN 1964 (A 627) 216 FREIGANG, SEIDEL and FLACH: 1968 (460) FREIREICH and LANDAU 1971 (A 24) 177 FREYSCHUSS and HOLMGREN 1965 (A 387) FRIBERG, NYSTROM and SWANBERG 1959 (573): 105, 102 FRITSCHE 1969 (461) 86, 85 FRONING, MATHER, DADDARIO and HARTUNG 1969 (A 592) 214: FRYZE, GRUSZKA and ZAWADZKI 1970 A 666) 219 FUKUI and KAKIUCHI 1970 (A 85) 181 GABRIEL 1969 (A 388): 201 GAENSHER, CADIGAN, ELLICOTT, JONES and MARKS 1957 (44) 17, 14; (149) 34, 33 GARLAND and PEARCE 1967 (A 481) 207 GARREL, PERRET, PELLAT and ARNOULD 1970 (A 163) 207 GARREL, PERRET, PELLAT and ARNOULD 1970 (A 482) 207 GASSMAN and WRANNE 1967 (A 25) 177 GATTO, CANEDA, CAVALLO and MASSIMILIA

1967 (A 389): 201

GAULTIER, FOURNIER, GERVAIS and BODIN 1964 (A 484) 207 GAULTIER, FOURNIER, GERVAIS, EFTHYMIOU, SRAER, DISMUTH, BODIN, CHRISTOFOROV, SICOT and FREJAVILLE 1968: (A 746) 226 GAULTIER, FREJAVILLE, BISMUTH and PEBAY-PEYROULA 1970 (A 628) 216 GAUME, BARTEK and ROSTAMI 1971 (508) 94, 93 GEDDES and STEINHARDT 1968 (A 86) 181 GEIER, TUTTIN, PASQUIER, NAJMAN and BOUR 1966 (532) 97, 96 GEMZELL, ROBBE and STROM 1958 (574) 105, 102 GEORGE and SCHEJTER 1964: (A 87) 181 GERBER and SEEWALD 1966 (A 747) 226 GERHARDT, GOTHERT, MALORNY and WILKE 1970 (A 390) 201 GERHARDT, GOTHERT, MALORNY and WILKE 1971 (A 806) 230 GERITSEN: 1964 (A 701) 222 GETTLER and MATTICE 1933 (45) 17 GIBBONS and MITROPOULOS 1972 (372) 72, 70 GIBSON, HELLER and YAKULIS 1966 (A 89) 181 GIBSON and KAMEN: 1966 (A 90) 181 GIBSON, PALMER and WHARTON 1965 (A 88) 181 GIBSON and PARKHURST 1968 (A 91) 182 GIEL 1965 (660) 115, 163 GIEVER 1967 (9) 9, 6 GIEVER and RUCH 1971 (A2 53) 192 GILL 1971 (A 667) 219 GIRARDI, CIS and PLATTI 1967 (A 485) 207 GIROND 1967 (575) 105, 102 GLADYSHEVSKAIA, DOLOSHITSKY and SOBCHUK 1966 (619) 112, 111 GLASEL 1970 (A 748) 226 GLASS, MALLACH and WOJAHN 1966 (A668) 219 GLASS, EDWARDS, DeGARRETA and CLARK 1969 (A 92) 182 GLASS, GARRETA, LEWIS, GRAMMATICOS and SZUR 1968 (409) 77, 74 GLASS, JACOBY, WESTERMAN, CLARK, ARNOT and DIXON 1968 (576) 105, 102 GLOWACKI, GRUDZINSKA and WACLAWIK 1958 - **(**432) 80, 79 GOKIN 1971 (283) 59, 57 GOLACKA, JANIK-KURYLCIO and ROZEK 1969 (A 669) 219 GOLDBERG and CHAPPELL 1967 (509) 94, 93 GOLDSMITH 1964 (10) 9, 6 GOLDSMITH 1967 (A: 254): 192 GOLDSNITH 1967 (A 670) 219 GOLDSMITH 1969 (661) 115, 164 GOLDSMITH 1970 (320) 64, 61 GOLDSMITH 1972 (662) 115, 165

GOLDSMITH and COHEN 1969 (II) 9, 7 GOLDSMITH and DEANE 1965 (A 286) 194 GOLDSMITH and LANDAW 1968 (12) 9, 6 GOLDSMITH and ROGERS 1959 (285): 194 GOLDSMITH, SCHUETTE and NOVICK 1963 (46) 17, 13, 14, (150) 34, 33 GOLDSMITH and TERZAGHI 1963 (76) 22, 21; (151) 34, 33 GOLDSMITH, TERZAGHI and HACKNEY 1963 (180) 40, 38 GOLDSTEIN 1965 (577) 105, 102 GOLDSTEIN and EPSTEIN 1972 (663): 115, 166 GORALSKI and JANUSZKO 1968: (A 486): 207. GORDON 1965 (A 487) 207 GORODINSKY, LEVINSKY and SCHERBAKOV 1967 (A 334) 198 GORSKI 1962 (284) 57, 59 GOTHE, FRISTEDT, SUNDELL, KOLMODIN EHRNER-SAMUEL and GOTHE 1969 (181) 40, 38, 39 GOTHERT, LUTZ and MALORNY 1970 (338) 67, 66 GOTO: 1968: (A 749): 226 GOULDING 1965 (A 629) 216 GOULDING 1967 (A 750) 226 GOULON, BAROIS, RAPIN, NOUAILHAT, AUGUSTIN, HENNETIER, BAGUET, KUNTZIGER and BRETEAU 1965 (A 751) 226 GOULON, BAROIS, RAPIN, NOUAILHAT, GROSBUIS and LARROUSSE 1969 (A 809) 230 GOULON, BAROIS, GAJDOS, LABROUSSE, SCHORTGEN, AMERONGEN and ROBERT 1970 (A 752) 226 GOULON and HENNETIER 1966 AA 807) 230 GOULON, LEVY-ALCOVER, NOUALHAT and DORDAIN 1967 (A 808) 230 GOULON, LEVY-ALCOVER, NOUAILHAT and DORDAIN 1968 (A 810) 230 GRAHAM and HITCHENS 1968 (A 630) 216-GRAMER and BECKENKAMPH 1966 (A 811) 230 GRAMER and ROUF 1968 (410) 77, 74 GRAZIANI and PAGGI 1965 (A 812) 230 GRAZIANI, ROSSI, CASTELLINO and SILVERSTRONI 1957 (243) 53, 52 GRAY and GIBSON 1970 (A 93) 182 GRAY and GIBSON: 1971 (A 94) 182 GRAY and GIBSON: 1971: (A 95): 182 GRAYBIEL 1942 (242) 53, 51 GREGORY, MALINOSKI and SHARP 1969. (A 631): 216 GREMY, SALMON, FRANCOIS and BERTIN 1968 (A 671) 219 GRIFFIN and HOLLOCHER 1967 (A 96) 182 GRIFFITHS 1970 (A 672) 219 GROB 1968: (119) 30, 29 GROHME, SCHNEIDER and MASSHOFF 1969. (533) 97, 96; (A 488) 207 GROSSE and NEUHAUS 1970 (552) 101, 99 GRUNDY 1969 (A 255) 192 GRUT, ASTRUP, CHALLEN and GERHARDSSON: 1970 (13) 9, 7

GUEPIN 1969 (A 702) 223

GUEST, DUNCAN and LAWTHER 1970 (462) 86, 85 GUEYE, BA and DIOP 1965 (A 673) 219 GUIDOTTI and KONIGSBERG 1964 (A 97) 182: GUILLERM, BADRE and GAUTIER 1967 (285): 59, 56 GULERIA, PANDE, SEITHI and ROY 1971. (A 391) 201 GULZOW 1957 (A 392) 201 GUNTHER 1971 (A 489) 207 GUTENKAUF, BRATT and ELIOT 1967 77, 75 GUY, SALHANY and ELIOT 1971 (412) 77, 75 GUYATT, NEWMAN, CINKOTAL, PALMER and THOMSON 1965 (A 393) 202. GYDELL 1966 (A 26) 177 HAAB and PIPER 1968 (A 394) 202 HAAGEN-SMIT 1966 (A 287) 194 HADDON, NESBITT and GARCIA 1961 (578): 105, 104 HAEBISCH 1970 (120) 30, 28 HAGGARD and GREENBERG 1934 (601) 110, HAHN and COPELAND 1966 (A 593): 214 HAKIM 1970 (580) 106, 102 HALL 1970 (463) 86, 85 HALL 1972 (433) 80, 79 HALPERIN, McFARLAND, NIVEN and ROUGHTON 1959 (440) 83, 81 HAMEL-PUSKARIC, BERITIC, JUSIC and FRANJIC 1970: (A:490):207 HAMILL and O'NEILL 1969 (77) 22, 21 HAMILTON and KERSTING 1970 (A 395) 202 HAMM 1966 (A 396) 202 HANISCH 1969 (A 98) 182 HANKE and KIERES 1967 (602) 110, 108: HANKS 1970 (483) 91, 89 HANQUET and LAMY 1971 (A 813) 230 HANSEN 1970: (A 491) 207 HANSEN, WILKE, MALORNY and GOTHERT 1972 (47) 17, 15; (153) 34 HANSON and HASTINGS 1933 (78) 22 34, 33 HANSSON and SUNDSTROM: 1969 (A 27) HANSZ and STYPEREK 1968: (464): 86, 85 HARADA and KOZUMA 1968 (534) 97, 96 (A 492) 207 HARADA, TSUKAYAMA, MIMURA, MINAMI and TATETSU 1971 (535) 97, 96 HARDING, WONG and NELSON 1964 (603) 110, 108 HARKE 1970 (95) 26, 24, 25 HARKE 1971 (100) 26, 25 HARKE and DREWS 1968: (121) 30; 29 HARTRIDGE 1920 (#8): 17, 11, 14 HATZFELD, WIENER and BRISCOE 1967. (A 397) 202 HAYASIINI, MOTOKAWA and KIKUCIII

1005051303

1966 (A 99) 182

AND ASTRUP 1966 (A 28) 177 HELLUNG-LARSEN, LAURSEN, KJELDSEN and ASTRUP 1968 (374) 72, 70 HELMCHEN and KUNKEL 1964 (441) 83, 81 HERNANDEZ, MAZEL and GILLETTE 1967 (A 556) 211 HERON 1962 (581) 106, 102, 104 HESS 1971 (101) 26, 25 HEXTER and GOLDSMITH 1971 (A 256): 1921 HEYDENREICH 1970 (442) 83, 81 HEYNDRICKX, SCHEIRIS, VERCRUYSSE and OKKERSE 1970 (A 753): 226 HILDEBRANDT, FRANKLIN, ROOTS and ESTABROOK 1971 (A 100): 182 HILPERT 1971 (A 398): 202 HIRANO, INOUE and TANAMI 1967 (604) 110, 109 HIRANO, INOUE and TANAMI 1968 (605) 2 110, 108 HIRANO, INOUE and TANAMI 1971 (A 814) HIRATA, HIOKI and HASHIMOTO 1969 (510) 94, 93 HIRSCH 1968 (A.594) 214 HLAVICA, KIESE, LANGE and MOR. 1969 (A 557) 211 HOCHSTRATE and OBERDISSE 1970 (A 558) 211 HODY and BAILEY 1968 (A 171) 187 HOFREUTER, CATCOTT and XINTARAS 1962 (49) 17, 15; (154) 34, 33 HOLCZABEK 1971 (286): 59, 58 HOLETON 1971 (A 595) 214 HOLETON: 1971 (A: 596) 214 HOLLAND 1965 (A 101) 182 HOLLAND 1967 (A 102) 182 HOLLAND 1969 (A 104) 182 HOLLAND 1969 (A 103): 182 HOLLAND 1970 (A 105) 182 HOLM 1950 (287) 59, 57 HOLMGREN: 1965 (A 402): 202 HOLMGREN 1965 (A 399) 202 HOLMGREN 1965 (A 400) 202 HOLMGREN 1965 (A 401): 202 HORIE 1964: (A 106) 182 HORIE 1965 (A 107) 182

HORVATH, DAHMS and O'HANLON 1971

HOWSE and SEDDON 1966 (620) 112, 111

HSI-PU and Li-MING 1910 (79) 22, 21

HSIEH, ROSS, SMALL and THOMPSON 1968

(484): 91, 89

(A 403) 202

HOSKO 1970 (443) 83, 81

HAYES and HALL 1964 (244) 54, 52

HEGGLIN 1944 (245) 54, 52

67, 66

and MOHLER 1972 (321) 64, 61

HAYWOOD, WALBERG, KERR, MOHSENIN

HEIDRICH and KLEMS 1969 (373) 72, 69

HEISTAD and WHEELER 1972 (340) 67, 66

HEIDRICH, BARCKOW and FRISIUS 1970 (339)

HELLUNG-LARSEN, KJELDSEN, MELLEMGAARD

Page 24U · HUBER, EPP and FORMANEK 1970. (A 1 08) 182 HUBERT 1943 (322) 64, 63 HUEPER 1944 (375): 72, 70 HUGHES and FISHER 1965 (A 674) 219 HUNDT and GRUNBERG 1960 (246) 54, 52 HYDE, MARIN, RYNES, KARREMAN 1971 (A 404) 202 IABLOCHKIN 1966 (A 335) 198 ICHIKAWA, HAGIHARA and YAMANO 1967 (A 559) 2111 IKEDA 1969 (444) 83, 81 IKUTA 1969 (A 494) 207 INANAGA 1966 (A 497) 208 INANAGA 1968 (A 498) 208 INANAGA, KUHARA, KUWAHARA. TORISU and SUZUKI 1966A (536) 97, 96 INANAGA, KUHARA, KUWAHARA and OGATA 1966B (537) 97, 96 INGIULLA, GRASSO and MARIOTTINI 1968 (A 288) 194 IPPEN and GOERZ 1969 (621) 112, 111 ISHIKAWA 1969 (A 499) 208 IWANOWSKA 1967 (A 815): 230 JACOBSEN 1971 (A 816) 230 JAFFE 1968 (A 190): 189 JAFFE 1970 (A 191) 189 JAFFE 1965 (288) 59; 57 JAFFE 1968 (323) 64, 61 JAGI and ZIMMERMANN 1934 (289) 59, 56 JAMES and RUMBLE 1967 (A 405) 202 JARRELL 1965 (122) 30, 28 JAVANOVIC and POLOVINA 1964 (A 29) 177 JECH 1972 (14) 9, 6 JEDRYCHOWSKI, KUS, PIOTROWSKI and SAWICKI 1965 (A 560) 212 JEDRYCHOWSKI, KUS, PIOTROWSKI and SAWICKI 1965 (A 561) 212 JEFCOATE and GAYLOR 1969 (A 109): 182 JENEY and MEDVE 1967 (A 597) 214 JESCHECK 1967 (A 500) 208 JOELS and NEIL 1962 (216) 49, 47 JOHNSON, DWORETZKY and HELLER 1968: JOHNSON and MILLER 1968 (A 408) 202 JOHNSON, TAYLOR and De GRAFF 1965 (A 407) 202 JOHNSON, TAYLOR and LAWSON 1965 (A 406) 202 JOHNSTON and BURGER 1971 (A 172) 187 JONES, YANT and BERGER 1923 (102) 26, 24 JONES, STRICKLAND, STUNKARD and SIEGEL 1971 (418) 77, 75 JONGBLOED 1939 (80) 22, 19 JOPKIEWICZ, KONECKI and WENTKOWSKI 1965 (622) 112, 111 JORDI 1967: (A 501) 208 JORDI 1968 (A 7.03) 223

JUDD: 1971 (A 173): 187

JUNGE, SEILER, BROCK, GREESE and RADLER 1971 (a 192) 189 KALIAEVA 1951 (290) 60, 56 KAMATAKI and KITAGAWA 1971 (A 562) 212 KAMPFFMEYER and KIESE 1965 (A 563): 212 KAMRAJ-MAZURKIEWICZ 1967 (581) 106, 102 KANAZIRSKY 1965 (A 409) 202 KARACAN, BARNARD and WILLIAMS 1971 **(5**38) 97, 96 KASHIMA, FUKUI, MASUDA, WAKASUGI and HAYAMA 1969 (675) 219 KATSUKI 1965 (A 502) 208 KATSUKI 1966 (606) 110, 108 KATSURA 1971 (445) 83, 82 KATZSCHMANN 1970 (291) 60,56 KAWAMOTO 1966 (A 410): 202 KAWAMURA 1971 (465) 86, 85 KAYES 1965 (14a) 8, 6 KAYES 1970: (A 632): 216 KAYSER 1939 (341) 67, 66 KEHL and KEHL 1967 (A 503) 208: -KEITH and TESH 1965 (123) 30, 29 KELLS 1968 (582) 106, 102 KEMKES 1941 (A 704) 223 KENT 1970 (A 174) 187. KERTESZ, ANTONINI, BRUNORI, WYMAN and ZITO 1965 (A 175): 187 KEYES, MIZUKAMI and LUMRY 1967 (A 110) KHACHATURYAN, MITAREVSKAYA and EGORENKOVA 1969 (511) 94, 93 KHROLENKO 1969 (A 504) 208 KILLICK 1940 (15) 9, 6 KILLICK and MARCHANT 1965 (A 754) 226 KIM and PARK 1968: (A-633): 216-KIM and RYO 1966 (A 336) 198-KIRIACHKO: 1966: (217): 49, 48 KITTEL and THEISSING-ERLANGEN 1968 (466) 86, 85 KITTREDGE 1971 (A 676) 219 KJELDSEN 1969 (376): 73, 69 KJELDSEN 1970a (377) 73, 70 KJELDSEN 1970b (378) 73, 70 KJELDSEN, ASTRUP and WANSTRUP 1972 (37.9) 73, 70 KJELDSEN and DAMGAARD 1968 (380):73, 70 KJELDSEN and DANIGAARD 1968 (414) 77, 74 KJELDSEN and MOZES 1969 (381) 73, 69. KLAUSEN, RASMUSSEN, GJELLEROD, MADSEN and PETERSEN 1968 (203) 46, 45, (292) 60, 56 KLAVIS and SCHULZ 1966 (293) 60, 57 KLEDECKI and WINIARSKI 1963 (247) 54, 52 KLINGENMAIER, BEHAR and SMITH 1969 (A 30) 177. KLINGHOFFER 1965 (A 677) 219 KOBUTNICZKY 1966 (553) 101, 99 KOCH 1965 (A 31) 177-KOELSCII 1936 (294) 60, 57 KOHN-ABREST 1949 (124): 30, 29

KOKAME and SHULER 1968 (A 817):230

KOLB 1968 (623) 112, 111

Page 241 KOLB 1968 (624) 112, 111; (A 505) 208 KOMURA 1967 (539) 97, 96 KONDRASHENKO, GLANTS and MAEROVICH 1971 (A 818) 230 KORNER 1965 (218) 49, 47 KOSMIDER, ZURKOWSKI and WEGIEL 1965 (A 111) 183 KOSTLER, OTTO, RITTIG and POLSTER 1967 (A 32) 177 KOSTYUKOVA 1951 (248) 54, 52 KOTTER, HUCH, STOTZ and PHPER 1969. (A 411) 203 KRAL, CERNOCHOVA and TUSL 1966 (A 412) 203 KRATOCHVIL, WILKS and GERRARD 1957 (485) 91, 89 KRATZ 1968 (A 564): 212 KRETSER 1964 (A 697) 222 KREUKNIET 1964 (A 413): 203 KREUZER and CAMPAGNE 1965 (413a) 203 KRISHMAN, KUPPUSWAMY, MANI and MAJID 1971 (A 678) 219 KROBER, LANGE, MATHIES and MOR 1968 (A 565) 212 KROBER, LANGE, MATHES and MOR 1970 (A 566) 212 KROETZ 1936 (295) 60, 56 KROETZ 1936a (324) 64, 63 KROETZ 1936b (325) 64, 63 KRUG 1965 (A 506) 208: KRUSMANN, SCHRODER and SCHRODER 1971 (A 33) 177 KRUSZYNSKI and HENRIKSEN 1969 (125) 30, 28 KUCHER and RIEDEL 1969 (A 819) 230 KUNZ, DONDES and HARTECK 1970 (A 337) 198 KUNTZMAN, LEVIN, JACOBSON and CONNEY 1968 (A 567) 212 KUPFER and WUNSCHER 1968 (512) 94, 93 KUROIWA 1967 (A679) 219 KUROIWA, MURAO, HARUMI, KATAYAMA, YAMAMOTO, CHEN and UEDA 1968 (249) 54, 52 KUROIWA, KATO and UMEZAKI 1968 (540) 97, 96 KUROIWA, SHIDA and KATO 1969 (A 507) 208 KUROIWA, SHIDA, NAGAMATSU, KATO and SANTA T 1967 (446) 83; 81 KUTTNER 1968 (467) 86, 85 LACHNIT 1964 (A 634) 216: LACOSTE 1971 (A 415) 203 LACOSTE and ROUCH 1966 (A 414): 203 LAMY and HANQUET 1968 (A 820) 230 LAMY and HANQUET 1969 (A 821) 231 LAMY and NOIRFALISE 1971 (A 680): 219 LANDAU, SMITH and LYNN 1969 (A 290): 195 LANDAW 1969 (A 222) 190 LANDAW 1970 (A 223) 191

LANDAW, CALLAHAN and SCHMID 1970

(A 112) 183

LINTON, ADAMS and LAWSON 1968

LIZANETS and ZARKEVICH 1971 (A 757)226

LOCKSIMN and BURRIS 1965 (A600) 215

LOGUE, ROSSE, SMITH, SALTZMAN and

LOEPER, VARAY and COTTET 1942

GUTTERMAN 1971 (A 226) 191

(555) 101, 99

(252): 54, 42

LITMAN 1968 (A 635) 217

LITZNER 1936 (342) 67, 66

LO COCO 1970 (A 419) 203

LOMONACO 1971 (A 177): 187

LONG 1969 (626) 112, 111 LARCAN, ROBERT, CALAMAI and FREJAVILLE LONGO 1970 (584) 106, 102 LONGO, POWER and FORSTER 1969 (586) LARSON, HAAG and SILVETTE 1961 (16) 106, 102 LONGO, POWER and FORSTER 1967 (585) LARSON and SILVETTE 1968 (17) 9, 7 106, 102 LARSON and SILVETTE 1971 (18) 9, 7 LOPEZ-MAJANO 1971 (A 420) 203 LATALSKI and PAWLOWSKA: 1969. (A.569): 212 LORENTE, VARELA and SEIJAS 1953 LATALSKI and PAWLOWSKA 1970 (A 570) 212 (253) 54, 52 LUDERITZ 1971 (182) 40, 38; (A 293) 195 LUNDEVALL 1972 (A 705) 223-LUOMANMAKI 1966 (A.157.) 186 LUOMANMAKI and COBURN: 1969 (A 158): 186 LAWTHER and COMMINS 1970 (18a) 9, 7; LUSTMAN and GEERTS 1971 (254) 54, 52 LYNCH and MOEDE 1972 (A 227) 191 LEAVELL and McINTYRE 1969 (625) 112, 111 Mac FARLAND; ROUGHTON, HALPERIN and LEBRETON and GARAT 1964 (A 292) 195-NIVEN 1944 (82) 22, 21 Lebreton and GARAT 1964 (A 681): 219 MACHATA 1968 (A 684) 220 MACKINTOSH: 1965. (A: 512), 208 LeBRETON and GARAT 1964 (A 682) 220 MacQUARRIE and GIBSON 1971 (A 115) MAENO and FEIGELSON 1968 (A 601): LEE and SCHRAUZER 1968 (A 598) 214 215 MAGDALENO 1968 (A 178): 187 -MAHRLEIN 1967 (A 422) 203 MAINARDI 1964 (255): 54, 52 MAISELS, PATHAK, NELSON, NATHAN and SMITH 1971 (A 228) 191 MA:LIK 1971 (A-636): 217-LEUCHTENBERGER, LEUCHTENBERGER and MAMATSASHVILLI 1970 (607) 110, 108 MANN 1965 (541) 97, 96 MANSLEY, STANBURY and LEMBERG: 1966 LEVIN, ALVARES and KUNTZMAN 1970 (A 114) (A 116): 183 MANTELL 1964 (587) 106 LEVIN and KUNTZMAN 1969 (A 572): 212 MANTZ and TEMPE 1968 (A 825) 231 LEVIN and KUNTZMAN 1969 (A 571) 212 MANTZ and TEMPE 1968 (A 826): 231 LEWEY and DRABKIN 1944 (251) 54, 52 MARANZANA 1964 (A 706) 223 LEWIS and BRINK 1966 (A 418) 203 MARCELET 1907 (129) 30, 29 MARCHIARO, MARGARIA, GAIDO and AQUARO 1964: (A 707), 223: LIKOFF, SEGAL and KASPARIAN 1967 (415) MARI and RIZZATTI 1964 (A 685) 220

MARKIEWICZ 1966 (A.38) 178

MARKIEWICZ 1967 (A 229) 191

MARKIEWICZ 1970 (103): 26, 24

95. 93

MARKS and SWIECICKI 1971 (513)

MATSUYAMA 1969 (588) 106, 102

MATTHEW 1970. (A 758) 226

MATTHEW 1970 (A 827) 231

MARLAND and BERSAY 1972 (326) 64, 61

LANDAW and WINCHELL 1966 (A 221): 190:

LANDAW and WINCHELL 1970 (A 224) 191

and KNOLLE 1969 (250) 54, 52

LANGMANN 1964 (A 257) 192

LAVERNE 1970 (486): 91, 88:

LAVERNE 1970 (A.755): 226

LAWSON 1970 (A 416) 203

LAWSON 1971 (A 113) 183

LAWSON 1972. (A 417) 203

LECLERQ 1970 (19) 10; 6

LEE 1908: (126): 30, 29

LEHR 1970 (A683) 220

LEVIN 1965 (A 756) 226

LEWIS 1967 (A 599) 214 LIGHTFOOT 1972 (A 176) 187

LILIENTHAL 1950 (20): 10, 6

LINDERHOLM 1965 (A 36) 178

LINDERHOM 1969 (A 225) 191

LINDQUIST 1970 (664) 115, 167

1966 (A 37) 178

LINCH and PFAFF 1971 (A 338): 198

LILIENTHAL and FUGUTT 1946 (447) 83, 81

LINDERHOLM, SJOSTRAND and SODERSTROM

183

77, 75

LEHMANN 1968 (A 35) 178

LEHMANN 1908 (127) 30, 27

LEHMANN 1909 (128): 30, 28

WEISS 1965 (81): 22, 20

LEONOWICZ 1967 (554): 101, 99

LEDINGHAM 1964 (A 823) 231 LEE 1966 (A 824) 231

(155) 34, 33

106

9. 7

- 1967 (A 822) 231

LANGAUER-LEWICKA 1966 (A 508) 208

LANGE, KASTNER and JUNG 1970 (A 568) 212

LANGMANN and KETTNER 1968: (A 291) 195

LAPRESLE and FARDEAU: 1966 (A 509): 208

LAPRESLE and FARDEAU 1967 (A 510) 208

LAPRLE and FARDEAU 1971 (A.511) 208

LARCAN, LANDES and VERT 1970 (583)

LANG, SCHUSTER, UNGERN-STERNBERG, HAUM-

Page 243

MATTHEW 1971 (A-637): 217 MATTHEW and PROUDFOOT 1965 (A 513) 208 MAUGII 1972 (A 193) 139. MAUNDERLY 1972: (A-121) 203 MAURER 1941 (343)[67, 66 MAURER 1941 (134) 80, 79 MAUTNER 1955 (382) 73, 69 MAWATARIS 1970 (514) 95, 93 MAZALESKI, COLEMAN, DUNCAN and NAU 1970 (A 573) 212 McBoy 1965 (A 686) 220: Mc CONNELL, DEAL and OGATA 1969 (A 117) 183 McCREDIE and JOSE 1967 (A:39) 178 McDOWELL 1971 (339): 198: Mc FARLAND: 1952 (187) 91, 88 McFARLAND 1970 (448) 83, 81 McFARLAND 1971 (A 17.9) 187 McFARLAND, ROUGHTON, HALPERIN and NIVEN 1944 (449) 83, 81 McFEE, LAVINE and SULLIVAN 1970 (A 340): 198 McGRATH and JAEGER 1971 (A 602) 215 Mc GRATH and MOFFA 1972 (A 603) 215 McILVAINE, NELSON and BARTLETT 1969 (50) 18, 15, (156) 34, 33 McMILLAN and COPE 1969 (A 194) 189 McNALLY 1931 (A 687) 220 MEDA 1964: (A 118): 183 MEDVEDOWSKY, SACCO and BELZUNCE 1965 (256) 54, 52 MEIGS 1948 (52) 18, 14; (157) 34, 33 MEIGS and RYAN 1971 (607) 110, 108 MELANOWSKI 1963 (450) 83, 82 MEL'NICHENKO 1968: (A 119) 183 MENKES, SERA, ROGERS, HYDE, FORRESTER and DuBOIS 1970 (A 423) 203 MENZ 1966 (A-688) 220 MERLI 1969 (A 180) 187 MESOLELLA, PERRELLA, TESTA and MORELLI 1970 (468) 86, 85 METCALFE, MALL, BARTELS, HILPERT and PARER: 1965 (589): 106, 102 MEYER, GROVER and WEIL 1972 (219) 49, 47 MIHAI and WEBER 1964: (344): 67, 66 MIKULPA 1970 (488) 91, 89 MILLER 1966 (A 828) 231 MILLS and EDWARDS 1968 (220): 49, 47 MIRANDA, KONOPINSKI and LARSEN 1967 (204): 46, 45 MIRKIN 1966 (A 294) 195 MISCHENKO and FRENKEL 1966 (515) 95, 93 MITCHELL and RENZETTI 1968 (A 424) 203 MITTMAN 1967 (A 425) 208-MIYAGISHI and HAYASHI 1968 (517) 95, 93 MIYAGISHI and SUWA 1969 (516) 95, 93 MIYAHARA and TAKAHASHI 1971 (A 230): 191 MOC. RICKARD and MOSS 1969 (A 604) 215 MOKHOV 1967 (A 341) 198 MOLFINO and ZANNINI 1964 (A 829) 231 MONACO 1964 (A 708) 223-

MONTGOMERY and RUBIN: 1971 (A 574) 213 MOON and RICHARDS 1972 (A 120) 183 MOORE and FINESTONE 1968 (A 514) 208 MORANDO and ROVIDA 1965 (183) 40, 38 MORGANSTERN, ASH and LYNCH 1970 (A 342) 198 MOROVIC: 1968 (A: 515) 208 MORRIS 1969 (469): 86, 85 MORRISON and HORIE 1965 (A 121): 183 MORROW 1967 (A 258) 192 MOSINGER, BISSCHOP and LUCCIONI 1969: (257) 54, 52 MOSS 1969 (A 709) 223 MOTLEY 1971 (52) 18, 15; (158) 35, 34 MOTTA 1940 (258) 54, 52 MOUNIER-KUHN, RICCHE, MORGAN and BERNAR 1968: (470): 86, 85 MOUNTAIN, CASSELL, WOLTER MOUNTAIN, DIAMOND and McCARROLL 1968 (184) MOUREN, POINSO, JOUGLARD, GIUDICELLIS. FRESCO and D'OMEZON: 1972 (627) 112, 111 MOUREU 1964 (185) 4, 38 MULHAUSEN, ASTRUP and KJELDSEN 1967 (382A) 73, 69 MULHAUSEN, ASTRUP and MELLEMGAARD 1968 (A 159) 186 MULLER and HUNG 1968 (A 689) 220 MULLER and VOIGT 1968 (416) 77, 74 MUMPOWER, LEWIS and TOUEY 1962 (130): 31, 29 MUROFUSCHI and MINAGAWA 1969. (A 690) 220, (A 516) 209. MURPHY and MULCAHY 1971 (590) 106, 103 MURPHY, LENG, ULRICH and DAVIS 1963 (A 295) 195

MURRAY 1971 (A 122) 183

1005051307

MONAUL 1940 (296) 60, 57

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NATIONAL ACADEMY OF ENGINEERING NATIONAL CLEARINGHOUSE FOR SMOKING NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH USPHS 1967 (23) 10, 8; (635) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH USPHS 1968 (24) 10, 8;(636) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH USPHS 1969 (25) 10, 8; (637) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH USPHS 1971 (26) 10, 8; (638) NATIONAL CLEARINGHOUSE FOR SMOKING AND HEALTH USPHS 1972 (27) 10, 8; (639) NATIONAL INSTITUTE FOR OCCUPATIONAL NISSARDI, SANNA-RANDACCIO, TORRAZZA NISSARDI, SANNA-RANDACCIO, TORRAZZA

NAGANO 1967 (A 123) 183 NAGEL 1937 (297) 60, 57

NAHUM 1965 (665) 115, 168

NAHUM 1968 (666) 115, 169

NAHUM 1969: (667): 115, 170:

NAKAO 1969 (608) 110, 109

1969 (21) 10. 7

114, 119

114, 121

114. 123

114, 128

NAGEL and GIBSON 1966 (A-124) 183

NAIRN, POWERM HYDE, FORSTER,

NASH and BEEBE 1969 (628) 112, 111

NATIONAL ACADEMY OF SCIENCES AND

AND HEALTH USPHS 1964 (22) 10, 8

SAFETY AND HEALTH 1972 (28) 10, 7

NECAS and NEUWIRT 1971 (518) 95, 93

NEWSOME and KEITH 1965 (181) 31, 29 NICOLAS and NICOLAS 1964 (556) 101, 99

NIDEN: and SCHULZ 1965 (221): 49, 47

NIELSEN 1971 (489) 91, 88, (A 759) 226

and CASCIU 1965 (A. 427): 204:

and GARIEL 1965 (A 428) 204

NOBEL and RICKER 1967 (A. 40) 178:

NISSARDI, TORRAZZA and ANEDDA 1967

NIZHEGORODOV and MARCHOZKY 1969 (A 575)

NOBLE, BRUNORI, WYMAN and ANTONINI

NOBLE, PARKHURST and GIBSON 1970 (A 128)

NISSARDI, SANNA-RANDACCIO and SARNA

NAVRATIL 1956 (345) 67, 66

NIEBROJ 1969 (451) 83, 82

NIELSEN 1971 (346) 67, 66

196S (A 430): 204

(A 429) 204

1967 (A 127) 183

213

184

NISHIGORI 1932 (347) 67, 66 NISHII 1968 (A 296) 195

NAGEL, GIBSON and HAMILTON 1971 (A 126) 183 NAGEL, GIBSON and JENKINS 1971 (A 125) 183

LAMBERTSEN and DICKSON 1965 (A 426) 203

NORMAN, DOUGLAS and SMITH 1966 (222) 49, 48 NORMAN and LEDINGHAM 1967 (A 760) 226 NORMAN, MacINTYRE, SHEARER and SMITH 1970 (A-830):231 NOUILHAT 1964 (A 761) 226 OBERSTEG and DELAY 1966 (691) 220 O'DONNELL, CHIKOS and THEODORE 1971a (490) 91, 89 O'DONNELL and MIKULKA 1970 (492) 91, 89 O'DONNELL, MIKULKA, HEINIG and THEODORE 1971b (491) 91, 89 OETTEL 1967 (104) 26, 24 OGATA 1968 (A 495) 207 OHARA 1968 (452): 83: 82 OHRESSER, CHASSON, JOUGLARD, GOUIN, DUBOULOZ and TASSY 1968 (A 831) 231 OKALYI 1969 (A 181):187 OKESON and DIVERTIE 1970 (223) 49, 47 OKULOVSKI and HACHATUROV 1968 (A 762) OKUMA, ISHINO, SUNAMI and MOTOIKE 1968 (A 517) 209 OKUNYEV and PROKHORENKO 1966 (519) **9**5, 93 OMURA and SATO 1964 (A 576) 213 OMURA, SATO, COOPER, ROSENTHAL and ESTABROOK 1965 (A 129) 184 ORINIUS 1968 (259) 54 ORIOLI and CATTANIA 1965 (542) 97, 96 ORIZAGA and DUCHARME 1967 (383) 73, 69 OSBORNE, ADAMEK and HOBBS 1956 (132) OSKI, GOTTLIEB, MILLER and DELIVORIA-PAPADOPOULOS 1970 (A 130) 184 OSTROWSKA 1967 (A 518) 209 OSWALD 1966 (A519) 209 OTIS 1970 (417): 77, 75 OTSUKA, FUJIWARA, IKAWA and HIRAYAMA 1970 (133) 31, 29 OTSUKI, NINOMIYA, YAMAMOTO, NAKASHIMA, SHUTARA, URAKAMI and ITAMI 1966 (A 763) OWEN and REYNOLDS 1967 (134) 31, 28 PAEZ 1970 (A 431) 204 PALADE, MIHAI, GOILAV and SOVAREL 1969 (A 520) 209 PALMA-CARLOS, PALMA-CARLOS and SOARES 1964 (A-41):178 PALMA-CARLOS, PALMA-CARLOS and SOARES 1966 (A 231):191 PAPAVASLIOU, COTZIAS, DUBY, STECK, FEHLING and BELL 1972 (A 764) 227 PARADE and FRANKE 1939 (260) 54, 51 PARE and YEUNG 1969 (520) 95, 93 PARIS 1964 (629) 112, 111

PARKHURST, GERACI and GIBSON 1970 (A 132)

PARKHURST and GIBSON 1967 (A 131) 184 POLITZER 1968 (A 138): 184 PARMEGGIANI and GILARDI 1952 (53) 18, 13, 14; (83) 22, 21; (159) 35, 33 PARROT, STUPFEL, ROMARY and MORDELET-DAMBRINE 1971 (224) 50; 47 PASECHNIK, SHTUMM, VLADISLAVLEV and ZAMAYATNIN 1971 (A 133) 184 PATTONO, MARCHIARO, CAPELLARO and ORIONE 1964 (A 160) 186 PATZ 1949 (261):55, 51 PAULEIKOFF, MULLER-FAHBUSCH, MESTER and MEIBNER: 1971 (A. 521); 209 PAULET and CHEVRIER 1966 (A 134) 184 PAULET and CHEVRIER 1969 (A 765) 227 PAULI, TRUNIGER, MARSEN and MULHAUSEN 1968a (557) 101. 99 PAULI, TRUNIGER, LARSEN and MULHAUSEN 1968ь (558) 101, 100 PEARCE 1968 (A. 522) 209: PECORA 1964 (A. 766) 227 PEDRERO and RODRIGO 1964 (A 347) 198. PERRELLI, PREVOT and SULOTTO 1970 (418) PERRELLI and ROSETTANI 1964 (A. 297) 195 PERRELLI, ROSETTANI and BRAGUZZI 1965 (A 2)8) 195 PETERSON, SIGGAARD-ANDERSEN, KRISTENSEN and KJELDSEN 1968 (348) 67, 66 PETERSON and STEWART 1970 (A 161) 186 PETIT, PETIT and GEILLE 1970 (A 692): 220 PETRILLI and KANITZ 1970 (186) 41, 38, 39 PETROVA, DALAKMANSKI and BAKALOV 1966 (A 299) 195 PETROVIC 1970 (419) 77, 75 PETTER, BOURBON, MALTIER and JOST 1971 (A: 135) 184 PETTY 1969 (A 832) 231 PHELPS and ANTONINI 1969 (A 136):184 PHILIPPE and HOBBS 1956 (135) 31, 29 PLACE 1970 (A 767): 227. PICKW.ELL 1970 (A: 195) 189. PIEDELIEVRE, BRETON and DEROBERT 1969 (591) 106, 102 PIERCE and COLLINS 1971 (A 343) 198-PINCHERUE and SHANKS 1967 (420) 77, 75 PIPER, PFEIFER and SCHEID 1969 (A 433) 204 PIPER and SIKAND 1966 (A 432) 204 PIRNAY, DEROANNE, DUJARDIN and PETIT 19716 (299) 60, 56 PIRNAY, DUJARDIN, DEROANNE and PETIT 1971a (298) 60, 56 PIRNAY, FASSOTTE, DEROANNE and PETIT

1968 (A 137) 184

PIRNAY, FASSOTTE, GAZON, DEROANNE

PIRNAY, PETIT and ROBERTS 1970 (A 435)

PODLESCH and STEVANOVIC 1966 (A 436): 204

and PETIT 1969 (A 434) 204

POGRUND 1969 (609) 110, 108

POLLARD 1970 (A 693) 220 PORTHEINE 1971 (105) 26, 25 POWER 1968 (A 162) 186 POWER, AOKI, LAWSON and GREGG 1971 (A 438) 204 POWER, MYDE, SEVER, HOPPIN and NAIRN 1965 (A 437) 204 PRELLWITZ, SCHUSTER, SCHYLLA, BAUM, SCHONBORN, UNGERN-STERNBERG; BRODERSEN and POEPLAU 1970 (421) 77, 74; (A. 638), 217 PREROVSKA and DRDKOVA 1967a (384) 73, 70 PREROVSKA and DRDKOVA 1967b (385) 73, 70 PREROVSKA and DRDKOVA 1971 (386) 73, 71 PREZIOSI, LINDENBERG, LEVY and CHRISTENSON 1970 (521) 95, 93 PROKOP and WABNITZ 1970 (453) 83, 81 PUKHOV 1964 (610):110, 108 PUKHOV 1965 (A 768) 227 PUREC and KRASNA 1967 (A 605) 215 QUINTANA, MIRETE and GARCIA 1969 (559) 101, 99 RADUSHVICH 1968 (A: 769) 227 RAMSEY 1972 (84): 22: 20 RAMSEY 1966 (A 300) 195 RAMSEY 1967 (A. 344) 198 RAMSEY 1969 (422) 77, 75 RAMSEY 1970 (188) 41, 37 RAMSEY 1972 (493) 91, 89 RAMSEY 1967 (187) 41, 38, 39 RANDOWA 1967 (A 440) 204 RANDOWA and SIERAWSKI 1964 (A 439) 204 RAPOPORT 1967 (A 770) 227 RAUSA, DIANA and PERIN 1968 (A 139) 184 RAUSA, PERIN and DIANA 1967 (A 300):195 RAY and ROCKWELL 1970 (494) 91, 89 RAY 1967 (A 606) 215 RAYFIELD 1967 (A 301) 195 RECKZEH and DONTENWILL 1970 (423): 77, 75 RECKZEH, RUCKER, HARKE and DONTENWILL 1969 (106) 26, 25 REDDEMANN, AMENDT and JAHRIG 1970 (A 639) 217 REED and TROTT 1971 (A 302) 195 REED 1970 (A 140) 184 RIECHEL, WOBITH and ULMER 1970 (189) 41, 37 REJSEK 1971 (A771) 227 REMMERS and MITHOEFER 1969 (A 441) 204 REPLOH, KLOSTERKOTTER and EINCK-ROSSKAMP 1966 (A 710) 223 REVOL, MONIER, COURJON, FOURMET and GERIN 1966 (543) 97, 96 REVSIN and BRODIE 1969 (A 607) 215 RHODES 1971 (225) 50, 48 RICCI, CAPELLARO and GAIDO 1964 (424) 77, 74 RI 1966 (630) 112, 111 RIKANS and VAN DYKE 1971 (A 577) 213 RINGEL and KLAWANS 1972 (A 523) 209. RINGOLD, GOLDSMITH, HELWIG, FINN and

SCHUETTE 1962 (85) 23, 19

RISPLER and ROSS 1965 (A 303) 195 RITTER 1956 (300) 601 57 RITUCCI and LUV ONI 1965 (A 694) 220 RIZZI 1968 (A 772) 227 ROBBINS, BORG and ROBINSON 1968 (A 345) ROBIN, RAVENS and BING 1969 (327) 65, 61 ROBINSON and ROBBINS 1970 (A 196) 189 ROCHE, BERTOYE, VINCENT, MOTIN, GARIN, BOLOT and CHADENSON: 1968 (A 633): 231 RODKEY 1970 (A 346) 198 RODKEY and COLLISON 1970 (A 42) 178 RODKEY, COLLISON and ENGEL 1969 (A 711) RODKEY, COLLISON and O'NEAL 1971 (A 43) 178; (A 182) 187 RONDIA 1970 (A 578) 213 RONDIA, GUYAUX and HEUSGHEM 1966 (A 304) 195 ROOT 1962 (29) 10, 6 ROPSCHITZ and OVENSTONE 1968 (A 640) 217 ROSE, JONES, JENKINS and SIEGEL 1970 (522) 95, 93 ROSE 1969 (A 695) 220 ROSE and ROSE 1971 (668) 115, 172 ROSENBERG 1968 (86) 23, 20 ROSENBERG 1971 (87) 23, 20 ROSENBERG 19.72 (88) 23, 20 ROSENBLUTH 1968 (A 524) 209 ROSENTHAL 1968 (A 696) 220 ROSKAMM 1964 (301) 60, 56 ROSSI-FANELLI and ANTONINI 1958 (631): 112, 111 ROSSIN and ROBERTS 1972 (30):10, 7 ROSSO and DUGHERA 1964 (A: 712) 223 ROUCH, RIOUFOL and BOURBON 1971 (54) 18, 13, 15; (160) 35, 33a ROUGHTON 1970 (A 141) 184 RUBINO 1964 (226) 50, 47 RUDOLPH, BOYLE, DRESDEN and GILL 1972 (632) 112, 11 RUEL and BAR DIE 1954 (55) 18, 14; (161) 35. 33 RUHL and LIN 1936 (56) 18; 14; (89) 23, 20, 21 RUMEN and CHANCE 1970 (A 142) 184 SADOKIERSKI 1965 (A. 713) 223: SAITA and LUSSANA 1971 (435) 80, 79 SALNIS and HACHATUROV 1970 (A 773) 227 SANDERS and WARRINGTON 1971 (A 525): 209. SANNA-RANDACCIO and NISSARDI 1969. (A 442) 205 SANZHIEVA 1970: (A 608) 215 SANZIHEVA and ZAVARZIN 1971: (A:609) 215 SARACOGLU 1951 (262) 55, 51 SARTORELLI 1967 (A 403) 205

SARUTA 1937 (136) 31, 29

1966 (544) 98, 96

SASAKI, HIRANO, NAGAHAMA and USUI

SATAKE, HDA, TATSUBANA, YAMAZAKI and MATSUOKA 1968 (A 444) 205 SATO 1966 (471) 86, 85 SATOH, KIYOTANI, MINAFI and KONDO 1966 (A 697) 220 SAVATEEV, TONKOPIJ and FROLOV 1970 (A 834) 231 SAYERS and DAVENPORT 1930 (31) 10, 6 SAYERS, YANT, LEVY and FULTON 1929 (495) 91, 88 SCHAEFER 1964 (A 183) 187 SCHAFFERNICHT, AIEGLER and REINHARD 1970 (A 714) 223 SCHIECHE, KEBLER and KOBER 1970 (A 715) 223 SCHIEVELBEIN 1968 (32) 10, 7 SCHIEVELBEIN and EBERHARDT 1972 (328) 65, 61 SCHLECHT 1971 (A 610) 215 SCHMELZER, STEINER, MAYER, NEDETZKA and FASOLD 1972 (A 143) 184 SCHMIDT 1970 (454) 83, 82 SCHMIDT 1971 (107): 26, 25 SCHMIDT 1939 (57) 18, 13, 14; (90) 23, 21; (162) 35, 33 SCHMIDT 1940 (58) 18, 14; (91) 23, 21 SCHOTT, TOMMASI, BOURRAT and MICHEL 1967 (A 526) 209 SCHRAUZER and LEE 1970 (A 611) 215 SCHRENK 1942 (59) 18, 13, 14 SCHULTE 1963 (496) 92, 88 SCHULTE 1965 (A 835) 231 SCHUTTMANN 1968 (A: 716): 223 SCOPPETTA 1968 (592) 106 SCORER 1971 (A 259) 192 SEGAL 1970 (523) 95, 93 SELING 1966 (263) 55, 52 SELTZER 1970 (669) 115, 174 SEMAR, TRESER and LANGE 1967 (A 348) 198-SESSA and SANNA 1966 (A 527) 209. SCASSELLATTI SFORZOLINI and SAVINO 1968 (137) 31, 29 SFORZOLINI and SAVINO 1970 (A 305) 196 SHAFER, SMILAY and MacMILLAN 1965 (264) 55, 52 SHAW, CINKOTAL and THOMSON 1966 (A 445) 205 SHIDA and KUROIWA 1969. (A. 528) 209 SHIELDS 1971 (92) 23, 20: SHIMOJIMA 1970 (A: 529): 209 SHINTANI 1968 (227) 50; 48 SHIRABE, MAWATARI and KUROIWA 1970 (A 717) 223 SHIRAKI 1969 (A 530) 209 SHIRUKI and TATETSU 1967 (A. 531): 209 SHOJI, YAMAMOTO, NISHIDA, ISHIKAWA, TAKADA and INOUE 1967 (A 306) 196 SIASEV 1966 (A 698) 220 SIEGEL and MOHLER 1969 (A 184) 188 SIEGENTHALER 1965 (A 232) 191 SIEGRIST 1966 (A 718) 224 SIEVERS, EDWARDS, MURRAY and SCHRENK 1942 (60) 18, 14 SIGGAARD-ANDERSEN, KJELDSEN, PETERSEN and ASTRUP 1967 (387) 73, 70

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SIGGARD- ANDERSON, PETERSEN, HANSEN and MELLEMGAARD 1968 (349) 67. 66 SIGGAARD-ANDERSEN, PETERSEN, HANSEN and MELLEMGAARD 1969 (350) 68, 66 SIKAND and PIPER 1966 (A4-16) 205 SILVER 1971 (A 307) 196 SILVERMAN and GARDNER: 1965 (A. 349): 198 SIMONE, REGGIAND and BET 1965 (A-447):205 SIMPSON and RITCHIE 1968 (A 836) 231 SIRS 1964 (A 144) 184 SJOSTRAND 1948 (436) 80, 79 SJOSTRAND 197.0: (A:233) 191 SLATER: 1950 (302): 60, 58 SLATER: 1967 (A 350): 198 SLUIJTER 1967 (A 837) 231 SMALL, RADFORD, FRAZIER, RODKEY and COLLISON 1971 (A 44) 178 SMITH 1965 (A 838): 231 SMITH and BRANDON: 1970 (A 699): 220 SMITH, BRIERLEY and BRANDON 1971 (A 532) 209 SMITH, DRYAN, FELDSTEIN, LEVADIE, MILLER, STEPHENS and WHITE 1970 (A 352) 199 SMITH, BRYAN, FELDSTEIN, LEVADIE, MILLER, STEPHENS and WHITE 1970 (A 353) 199 SMITH, BRYAN, FELDSTEIN, LEVADIE, MILLER, STEPHENS and WHITE 1970 (A 354) 199 SMITH, BRYAN, FELDSTEIN, LEVADIE, STEPHENS and WHITE 1970 (A 351) 198 SMITH, BRYAN, FELDSTEIN, LEVADIE, MILLER and WHITE 1972 (A 355) 199-SMITH 1966 (A 356) 199 SMOKING AND HEALTH 1964 (634) 114, 116 SNASHALL 1970 (A 774) 227 SNYDER 1970 (A 533) 209 SOBOTKA and SOBOTKA 1969 (303):60: 57: SOKOLOVSKY, PINCHUK and PRAVOVEROV 1967 (A 260) 192 SOLVSTEEN and KRISTJANSEN 1968 (388) 73, 69 SPINAZZOLA, MARRACCINI, DEVOTO and ZEDDA 1966 (A 302 196 SRCH 1967 (190) 41, 36, 38 STAMM 1967 (A: 357.) 199. STANKOVIC, KANTA, FOCO and ALJINOVIC 1964 (472) 86, 85 STATHERS, HAEGER-ARONSEN, JONSSON and MARCIC 1968 (234) 191 STEINER, FRAYSER and ROSS 1965 (A 448) STEINER, LARSEN, DONATH and PAULI 1971 (560) 101, 100 STEINMAN 1937 (265) 55, 51 STEMHAGEN 1959 (138) 31, 29 STEVENS 1968 (A 185) 188 STEWART, FISHER, HOSKO, PETERSON, BARETTA and DODD: 1972 (A 719): 224

STEWART, PETERSON, BARETTO, BACHAND, JOSKO and HERRMANN 1970 (197) 92, 89 STICHNOTH and ZUMBANSEN 1970 (A 670): 220 STOKINGER 1969: (A 261) 192 STOKOWSKI and KESIAK 1969 (593) 106, 102 STORMER 1938 (266) 55, 51 STRZELCZYK and ZENK 1964 (473) 87, 85 STUPFEL 1970 (A 236) 191 STUPFEL and BOULEY 1970 (525) 95, 93 STUPFEL, BOULEY, DEKOV, BOURGEOIS and ROUSSEL 1968 (524) 95, 93 STUPFEL, BOULEY and POLIANSKI 1970 (611) 110, 103 STUPFEL and GODIN 1969 (A 197) 189 STUPFEL and ROUSSEL 1968 (A 235) 191 STURNER 1971 (A 641): 217 SUCHCICKI 1970 (A 671) 221 SUGIMOTO and YASUMITSU 1969 (A 839) 231 SULOTTO, BONZANINO, MEO and RUBINO 1969 b (352) 68, 66 SULOTTO, MEO, POLI and RUBINO: 1969a (351): 68, 66 SUNDSTROM: 1970 (A 45) 178 SUNDSTROM: 1972 (A 46) 178 SUPFLE 1933 (A 720) 224 SUPFLE 1934 (353) 68, 66 SUZUKI 1969: (304) 60, 57: SWINNERTON 1971 (A 200) 189 SWINNERTON, LINNENBOM and LAMONTAGNE 1970 (A-199): 189: SWINNERTON, LINNENBOM and LAMONTAGNE 1970 (A-198) 189 SZADKOWSKI, MASTAŁL, SCHALLER and LEHNERT 1970 (191) 41, 38 SZILAGYI 1967 (594): 106, 102 SZLIWOWSKI and KLEES-DELANGE 1970 (455) 83, 81 SZOLLOSI, MEDVE and JENEY 1970 (329) 65, 61 SZUCHOVSZKY, KENYERES and HARSANYI 1969 (A: 642): 217 TABER and MORRISON: 1964 (A 612) 215 TACCOLA, JEDRYCHOWSKI and CAVALLERI 1965 (456) 84, 81 TAKAHATA and MIYAGISHI 1969. (527): 95, 98 TAKAMATSU, TAKEICHI and YUKITAKE 1969 (A 534) 209 TAKEYA, TAKANO; TAMURA, HOJO, YOSIDA and HURUKAWA 1970 (A 840) 232 TANAKA 1967 (595) 106, 102 TANIEWSKI and KUGLER 1964 A (474) TANIEWSKI and KUGLER 1964 B (475) 87, 85 TARTULIER, TOURNIAIRE, DEYRIEUX and BLUM: 1967 (A449) 205 TATEGAMI 1968 (A: 775) 227 TATETSU, HARADA, NAKAMURA, KASAGI, ISIKAWA and KAMANO: 1968 (547) 98, 96

TATETSU, KIYOTA, TOYA, TEROKA, FUJITA, INQUE, MIMURA, HARADA, TAKAGI, YAMAGATA, KOZUMA, MIYAGAWA, TOMONARI, TERAOKA, MURAYAMA, YASUOKA, MIYOSIII and KASAGI 1967 A (545) 98, 96 TATETSU, TOYA, FUJITA, INQUE, HARADA, TOMONARI, MURAYAMA _ YASUOKA, MIYOSIII and KASAGI 1967b **-98.** 96 TATETSU, TOYA, MIMURA, HARADA and TSUKAYAMA 1969 (A 721) 224 TAYLOR and MILLER 1965 (A 48) 178 TEBBENS and SPEAR 1971 (A 722) 224 TEICHNER 1967 (528) 95, 93 TEMMERMAN and ETIENNE 1969 (A 841) 232 THEODORE, O'DONNELL and BACK 1971 (425): 78, 75; (33): 10, 6 THIELS, DURME, VERMEIRE and PANNIER 1972 (267) 55, 52 THOMAS and PEARSE 1964 (528a) 95, 93 THOMSON 1971 (A 672) 221 THURSTON 1968: (A 723): 224 THURSTON 1968: (A 842): 232 THURSTON 1970 (A 776): 227 THURSTON 1971 (A 843): 232 TIBBLET 1971 (330) 65, 61 TIBBLING 1969 (476) 87, 85 TIMMONS: 1970 (A: 613) 215 TKACHENKO, TISHCHENKO, ZATSEPILIN and DIMITROVA 1966 (426) 78, 74 TLUSTY, HLOUSKOVA, KROFTA and DAUM 1972 (A 540) 205 TOKANA 1971 (A 47) 178 TOMASINI 1967 (A 535) 210 TOMONARI: 1968 (548) 98, 96 TONOMURA, YAMATE and TSUJI 1967. (A 262) 193 TORELLI 1964 (A 777) 227 TOTH: 1907 (139) 31, 29 TOTSUKA, MORO, HORIE and YZAKI 1971 (549): 98, 96; (A 673) 221 TOWNSEND and STETSON 1968 (A 778): 227. TOYA: 1967 (A: 674): 221 TOYAMA 1968 (A 263) 193 TRAKHTENBERG: 1966 (A 675): 221 TRILLET, GIRARD and BOULETRHAU 1970 (A 536) 210 TRINDER and HARPER 1962 (61) 18, 15; (163) 35, 33 TRINQUET, CLAUZEL, CARRE and MEYER: 1967 (A-451) 205. TRINQUET and MEYER 1971 (199) 14, 43 TROMPEO, TURLETTI and GIARRUSSO 1964 (A 309) 196 TRONZANO and COSCIA 1964 (A 537) 210 TROUTON and EYSENCK 1961 (497 A): 92, 88 TRUDINGER: 1970 (A 614): 215 TRUNAUT, BOUDENE and CLAUDE 1965 (612) 110, 108

TRUHAUT, BOUDENE and CLAUDE 1967 (A 163) 186 TRUHAUT, BOUDENE and CLAUDE: 1968 (389) 73, 70 TRUHAUT, BOUDENE and CLAUDE: 1968: (427) 78, 75 TRUHAUT, BOUDENE, CLAUDE, JACOTOT 1968 (354) 66, 66 TUTT 1970 (A 676) 221 TZAGOLOFF 1965 (A 145) 184 UBISCH and WESTERLUND 1971 (A 314) 196 UMEZAWA 1968 (A 310) 196 VALIC and DURIC 1954 (62) 18, 14; 164, 35, 33 VANDENBERGH, BILLIET, WOESTIJNE and GYSELEN 1968 (A 452); 205-VAN LIEW 1968 (356) 68, 66 VAN: LIEW 1968 (355) 68, 66 VAN LIEW 1970 (357) 68, 66 VANNESTE 1966 (A 358) 199 VANROUX and GREGOIRE 1964 (A 453): 205 VAN VUGT 1968 (428) 78, 74 VARESE and SORANZO: 1968 (A.643):217 VAUGHAN, JENNELLE and LEWIS 1969 (228) 50, 48 VEIL 1968 (A 311) 196 VEITH 1940 (305) 60, 58 VENGERSKAIA, NAZOROV, BOBROVA SUBROVSKY and DUMKO 1968 (A7.24224 VENNESLAND and JETSCHMANN 1971 (A 615) 215 VICH 1969 (A 677) 221 VIDAL and PICARD 1967 ((A 538): 210 VIEWEG, GRUNEWALD and ZIEGLER 1970 (A 644) 217 VIOLET and PERROT 1967 (A 678) 221 VIVOLI and PREITE 1966 (A 679) 221 VOGEL 1968 (A 645) 217 VOGEL, WHEELER and WHITTEN 1972 (307) VOGEL and GLESER 1972 (306): 60, 56 VON BERGMANN 1934 (268) 55, 52 VON!OETTINGEN, DONAHUE and VALAER 1941 (358) 68, 66 VOORHOEVE, REMEIKA, FREELAND and MATTHIAS 1972 (A 312) 196 VORONCHUCK: 1966 (A 680) 221 VOROSMARTI, BRADLEY, LINAWEAVER, KLECKNER and ARMSTRONG 1970 (A 186) 188 VUIA: 1967 (A 539): 210 VUOPALA, HUHTI, TAKKUNEN and HUIKKO 1970 (A 725) 224 VYSKOCIL 1956 (359) 68, 66 WAGNER 1964 (A 726) 224 WAGNER and RICHTER 1968: (A 779) 227. WAGNER, MAZZONE and WEST 1971 (A 454) 205 WAGNER, LATHAM, BRINKMAN and FILLEY 1969 (A 455) 205 WATIL 1899 (140) 31, 27 WAJGT 1971 (437) 80, 79

1005051312

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WALTZ and HAUSERMANN 1965 (141) 31, 28 WANSTRUP, KJELDSEN and ASTRUP 1969 (390) 73, 70 WARBURG, GEISSLER and LORENZ 1967 (A 579):213 WATABE 1969 (A 780):227 WATANABE, KITAGUCHI, KIYOFUJI, MORISAKI, MASUDA, NOGUCIII and MATSUMOTO 1970 (A 681) 221 WATSON 1968 (A 682) 221 WAYLAND and MOHAJER: 1971 (A 616) 215 WEASE 1967 (A 359) 199. WEATHERBURN and LOGAN 1969 (A 50): 178 WEAVER 1971: (A 313) 196 WEBER, MORET and CHAUVET 1967 " (A 456) 205 WEDERKINCH 1964: (A: 51) 178 WEIGT 1967 (A 683) 221 WEINSTOCK 1969 (A 201) 189 WEINSTOCK and NIKI 1972 (A 202):189 WEISKOPF and SEVERINGHAUS 1972 (A 457) WEISS, SLAWSKY and DESFORGES 1971 (63) 18, 15; (165) 35, 34 WENDER 1963 (A 540) 210 WENDT 1941 (269) 55, 51 WENNELLAND 1945 (64) 18, 14 WERNITSCH 1969 (A 844) 232 WESTBERG and COHEN 1971 (A 203) 189 WHARTON 1964 (A 146) 185 WHEREAT 1970 (391) 73, 70 WHITE 1970 (A 238):191 WHITE, COBURN, WILLIAMS, GOLDWEIN, ROTHER and SHAFER 1967 (A 237): 191 WHITEHEAD and WORTHINGTON 1961 (93) 23; (166) 35, 33 WHO CHRONICLE 1971 (A 268) 193 WHO EXPERT COMMITTEE 1963 (A 264) 193 WHO EXPERT COMMITTEE 1964 (A 265) 193 WHO EXPERT COMMITTEE 1969 (A 267) 193 WHO SCIENTIFIC GROUP 1968 (A 266) 193 WIECZOREK 1968 (A 52): 178 WIETHAUP 1968 (A 315) 196 WIJDEVELD: 1968: (561): 101; 99 WIKTOR 1954 (331) 65, 63 WILLI AMS 1964 (192) 41, 37 WILSON, RICH and MESSMA 1972 (A 684) WILSON and HARDING: 1970 (613):110; 108 WILSON, NELSON and HARDING 1965 (A 5801 213 WINTER and SHATIN 1970 (A 845) 232 WINTERHALTER, AMICONI and ANTONINI 1968 (A 147) 185 WITTENBERG, ANTONINI, BRUNORI, NOBLE WITTENBERG and WYMAN 1967 (A 148): 185 WITTENBERG, BRUNORI, ANTONINI,

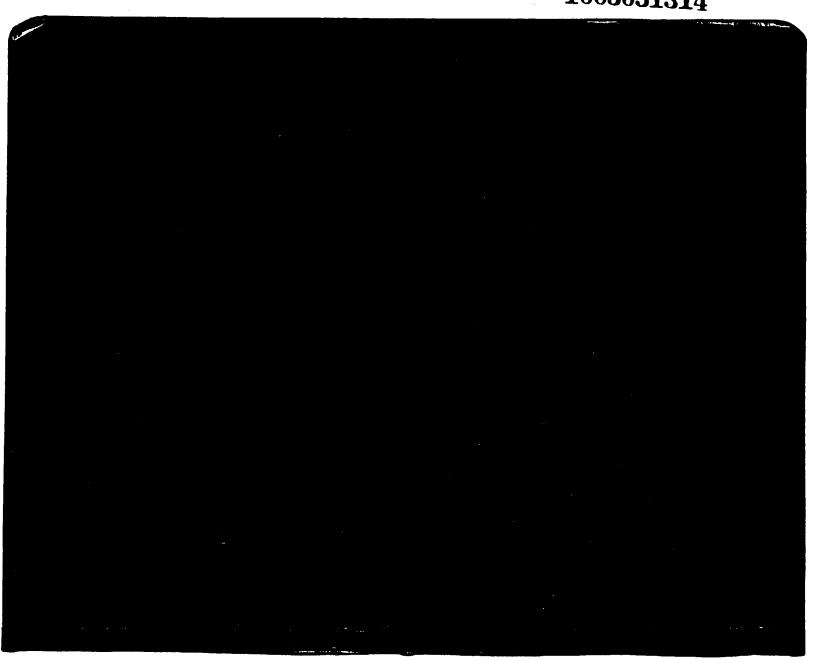
WITTENBERG and WYMAN: 1965 (633) 112,

WALD and FENTON 1972 (A 49) 17.8:

WITTGENS 1966 (614) 110, 108 WITUSIK 1971 (A 541): 210-WOHLERS, NEWSTEIN and DAUNIS 1967 (A 727) 224 WOHLRAB and OGUNMOLA 1971 (A 581) 213 WOJAHN: 1967 (A-53) 179: WOJCZUK and CHYLAK 1971 (562) 101, 99 WOLFGANG 1970: (A 204): 189: WOLKONSKY 1969. (A 269) 193; (A 458) 205 WOODRUFF 1970 (A 316) 196 WOOLF 1964 (A 459) 205 WRANNE 1967 (A 164) 186 WRANNE: 1967 (A239) 191 WRANNE 1969 (A 149) 185 WYNDER and HOFFMANN: 1967: (34) 10, 7 XINTARAS, JOHNSON, ULRICH, TERRILL and SOBECKI 1966 (A 360) 199 YACOUB, FAURE, MALLON and CAU 1970: (65) 18, 13, 15; (167) 35, 34 YAGLOU 1955 (108) 26, 24 YAMATE and MATSUMURA 1968 (206): YAMATE and MATSUMURA 1971 (A 317) 196 YAMAZAKI, OSHISHI and YAMAZAKI: 1970 (A 150) 185 YASUKOCHI and YASUOKA 1967 (A 542) 210 YASUOKA 1970 (550) 98, 96. YOUNG and PUGH 1963 1963 (598) 107, 104 YOUNOSZAI, KACIC and HAWORTH: 1968 (596) 106, 104 YOUNOSZAI, PELOSO and HAWORTH 1969 (597) 107, 103 YUKITAKE 1970 (A 543) 210 ZAFFIRI 1964 (A 781) 228 ZAFFIRI, CALA, CENTI and SALICONE 1971 (A 782) 228 ZANARDI, VILLA and MONTI 1964 (270): 55, 52 ZARIVAISKALA 1966 (A 728): 224 ZEH 1960 (332) 65, 63 ZENK 1964 (477) 87, 85 ZENK 1965 (478) 87, 85 ZIBEROV 1966 (A 783) 228: ZIEGLER 1936 (308) 60, 56 ZOLOTUKHIN 1968 (457): 84, 81 ZORN 1964 (A 685) 221 ZORN 1968 (A 846):232 ZORN 1969 (A 54) 179



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